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CONCERNING NEUROVASCULAR FACTORS IN PANCREATITIS*

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INTRODUCTION

As early as 1862, Panum,[9] a Copenhagen physiologist, suspected that vascular disturbances contribute to the pathogenesis of acute pancreatitis; he had observed that by injecting wax emboli into main stem pancreatic arteries, he could produce pancreatic hemorrhage. More recently, Block and co-workers,[2] in 1954, concluded, on the basis of studies they had made with rats, that a focal form of acute pancreatitis can result from prolonged, severe and extensive ischemia alone.

The possibility of such an ischemia resulting from nervous elements also has been considered and reported in the literature. In 1931, Beneke[1] theorized that reflex pancreatic ischemia, mediated by the celiac ganglion as a consequence of biliary ductal distention, could result in pancreatitis; no experimental data in support of such a view was presented. In 1956, Bossak and Joelson[4] were unable to histologically demonstrate sufficient involvement of pancreatic vessels to account for the striking occurrence of acute pancreatitis in a group of diabetic patients; hence, they suggested functional ischemia as an etiologic factor in the development of the disease among diabetics.

Whereas Ivy and Gibbs[7] have stated that there is no evidence to indicate that impairment of blood supply occurs with significant frequency as an initiating factor in the pathogenesis of acute pancreatitis, Blumenthal and Probstein[3] have pointed out that secondary vascular changes may be of ultimate importance as the essential link between a relatively trivial disease and a fulminating hemorrhagic process.

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AIM

Such a background of literature poses the question of whether neurovascular mechanisms are involved in the evolution of this disease. It is of particular interest to ascertain whether reflex parenchymal ischemia contributes to the propagation of pancreatitis from one area of the gland to another.

To demonstrate satisfactorily such a mechanism, it is felt that all other factors contributing to the spread of pancreatitis within the gland should be eliminated. This requirement can be satisfied, it is believed, by isolating a part of the pancreas from the remainder of the gland while retaining the peripheral autonomic innervation and the vascular continuity of both parts with the animal. With such a preparation, it is believed, any pancreatitis resulting in the isolated parenchymal segment as a consequence of pancreatitis experimentally produced in the *in situ* part of the gland will indicate a neurovascular mechanism of disease propagation.

METHODS

The dog was selected as the subject for this study. Previous unpublished investigations[5] on the pancreas in this animal have demonstrated a marked retention of pancreatic fetal characteristics. The pancreas of the dog is composed of two

distinct lobes with a narrow area of contact and a minimum of functional interdependence.

A ventral lobe is suspended in the mesoduodenum. Its duct joins the common bile duct before emptying into the duodenum. The blood supply of this lobe is derived largely from the superior mesenteric artery. At the most distal tip, a right pancreatic artery enters the parenchyma and supplies the gland to a point 2 or 3 inches away where it gains anastomosis with the pancreaticoduodenal vessels.

The dorsal lobe of the pancreas is drained by a duct entering the duodenum separately about 1 inch distal to, and slightly to the left of, the ventral duct aperture. The blood supply of this lobe is derived from the celiac axis, a left pancreatic artery coming from the splenic, and a middle pancreatic artery arising from the superior pancreaticoduodenal (fig. 1).

Richins[10] has shown that the nerve supply to the pancreas accompanies the servicing arteries, fibers from the celiac ganglion following the course of the branches of the celiac axis, and fibers from the semilunar ganglion taking the pathway of the vessels arising from the superior mesenteric artery. It seems reasonable, therefore, to assume that if the major servicing arteries are left intact, the concommitant autonomic innervation of the pancreas will be undisturbed.

Transecting the ventral lobe of the gland just distal to its penetration by the tributary from the marginal pancreaticoduodenal artery and freeing the mesenteric anchorage of the distal segment so formed down to the origin of the right pancreatic vessel effectively mobilizes a portion of the pancreas which may be delivered through the anterior abdominal wall. Closing the muscles behind this segment and suturing the skin over it constitute an isolated pancreatic segment which still retains its neurovascular continuity by the pedicle of mesentery extending from its distal tip.

This procedure is accomplished by teasing parenchymal lobules from one another along a developing plane of cleavage until the two parts of the ventral lobe are connected by two or three vascular bundles and by the duct. Vessels are divided between ligatures, and the proximal section of the duct is ligated. The distal duct is left open to drain freely through the skin as a small pancreatic cutaneous fistula.

In order to produce acute pancreatitis in the

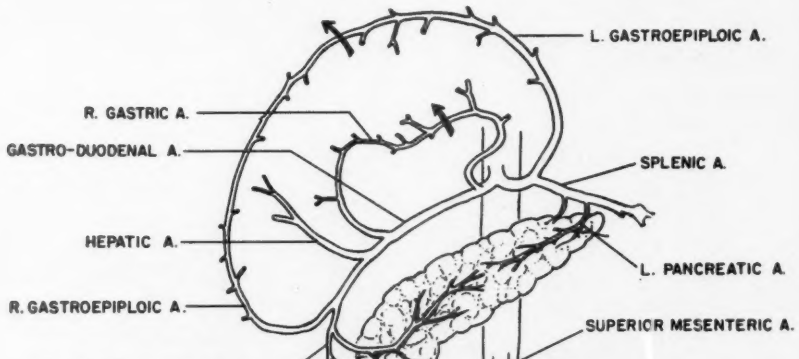

FIG. 1. Shown here is a diagrammatic representation of the arterial supply for the canine pancreas. The area chosen for ventral lobe transection is demonstrated, and the resulting isolated parenchymal segment is reflected to the left on its right pancreatic artery. *Arrows* indicate directions of reflections used for the sake of simplicity.

FIG. 2. The *top* specimen in the illustration is the complete dorsal lobe and portion of the ventral lobe 24 hours following bile injection of the dorsal lobe duct. Note the tremendous edema and hemorrhagic necrosis. The smaller *bottom* specimen is the isolated segment of the ventral pancreatic lobe from the same animal. Slight edema and parenchymal hemorrhage are apparent on the blunt extremity where transection was performed.

in situ parenchyma, sterile bile is aspirated from the individual dog's gall bladder and injected into the duct of the dorsal lobe, the aperture of which is exposed by duodenotomy. The injection is performed through a polyethylene cannula which is ligated in the duct, exerting sufficient pressure to force the green bile into the parenchyma.

The use of bile injection to produce pancreatitis is inspired by the work of Thal.[11] He has concluded from his research with bile pancreatitis that the interstitial injection of bile causes extensive and prolonged circulatory stasis within the pancreatic parenchyma both by direct action on the capillary wall and by spasm of arteriolar muscle. Hence, it seems that a neurovascular mechanism might be involved in this type of experimental pancreatitis.

CONTROLS

In order to understand the results of this procedure, two sets of controls are used. In the first, pancreatic transection and ventral lobe isolation are executed without bile injection of the dorsal lobe duct. In the second set of controls, bile injection of the dorsal duct without pancreatic transection is performed.

RESULTS

To qualify the experimental data, criteria for a diagnosis of acute pancreatitis require the presence of gross and microscopic hemorrhagic necrosis in the glandular substance. The presence of pancreatic edema and fat necrosis of adjacent tissues is unacceptable as a part of this standard. The operative procedure is sufficiently traumatic to produce tissue edema in the region of parenchymal transection, and the drainage of pancreatic ferments into the subcutaneous tissues surrounding the isolated ventral segment is expected to result in varying degrees of fat necrosis.

In 5 animals submitted to pancreatic transection and ventral segment isolation, no evidence of pancreatitis was demonstrated in either part of the gland. The 5 animals were alive 4 months after this operation, with pancreatic juices draining from their small cutaneous fistulas.

FIG. 3. Microscopic section from the *top* specimen in figure 2 shows edema, parenchymal hemorrhage and necrosis, typical of acute hemorrhagic pancreatitis.

In the 5 animals undergoing dorsal ductal bile injection without ventral segment isolation, the entire pancreas was involved in an acute pancreatitis. These animals expired within 24 hours.

Five dogs subjected to ventral lobe isolation and dorsal ductal bile injection survived from 24 to 48 hours. In these animals, the isolated ventral segment appeared both grossly and microscopically normal, except in the area immediately adjacent to the plane of transection where subcapsular hemorrhage and edema were evident. In every instance, however, the *in situ* pancreas was consumed in a hemorrhagic, necrotic process (figs. 2 to 4).

DISCUSSION

This study has demonstrated no neurovascular mechanism of disease propagation in bile pancreatitis. However, in the light of recent work presented by Honjin[6] and by Kuntz and Napolitano,[3] the possibility of neurovascular reflex ischemia has not altogether been eliminated. These observers have been able to demonstrate histologically a terminal neurofibrillar network appearing to consist of a syncytial protoplasmic structure. No terminal nerve endings are seen, suggesting that both sympathetic and parasympathetic innervations terminate in a common final protoplasmic network which encompasses the entire gland. The functional aspects of this syncytium have not been clarified, but it is possible that such a network may be involved in the induction of a spreading wave of pancreatic ischemia as a result of a localized trauma.

Inasmuch as tissue isolation protects against the spread of pancreatitis from diseased to normal parenchyma, it is concluded that tissue continuity seems to be necessary for such a process

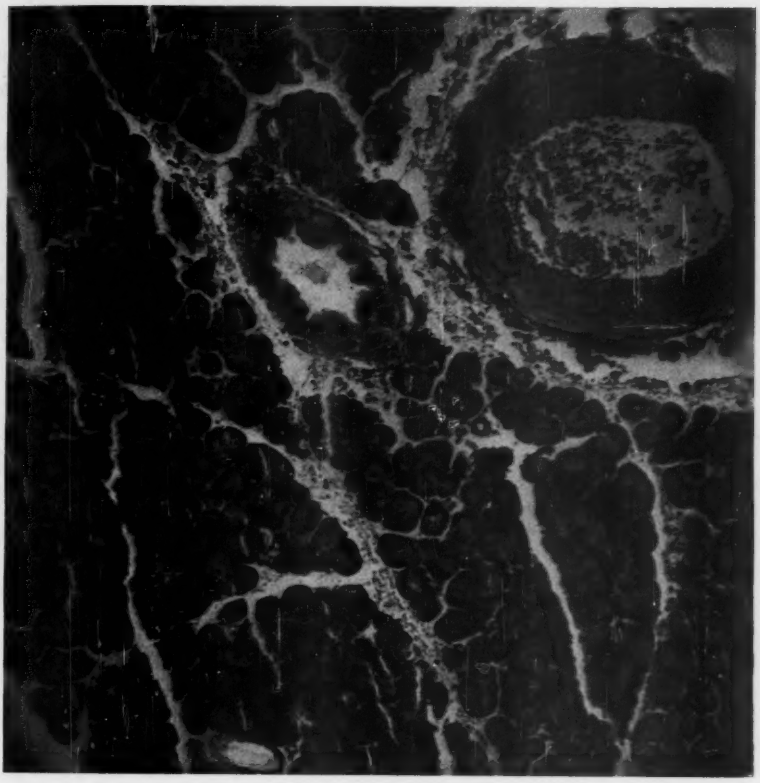

FIG. 4. Microphotograph is taken from the isolated ventral lobe shown as the *bottom* specimen in figure 2. Note the absence of edema, hemorrhage and necrosis.

of spread to occur. This study, while not demonstrating the mode whereby such a propagation of pancreatitis transpires, has suggested that peripheral neurovascular mechanisms do not appear to be involved.

SUMMARY

1. The literature has suggested that a neurovascular mechanism may be involved in the pathogenesis of acute pancreatitis.

2. The injection of bile into the dorsal pancreatic duct in five dogs resulted in acute pancreatitis involving both dorsal and ventral pancreatic lobes.

3. Transection of the pancreas with isolation of a segment of its ventral lobe produced no pancreatitis in five additional dogs.

4. Transection of the pancreas with isolation of a segment of its ventral lobe followed by bile injection of the dorsal pancreatic duct resulted in acute pancreatitis in the dorsal lobe, but no disease appeared in the isolated parenchymal segment.

5. It has been concluded from this study that tissue continuity is necessary for the spread of pancreatitis from one area to another of the parenchyma.

6. No neurovascular mechanism involved in the spread of acute pancreatitis could be demonstrated by this study.

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CARDIAC ARREST

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Cardiac arrest during surgery is said to occur in approximately one case in 2000 in the United States. The catastrophe probably occurs between 8000 and 9000 times each year in this country.

We have been involved with, or been aware of, an occasional cardiac arrest in the operating rooms at St. Anthony Hospital in Oklahoma City. This paper is the result of our concern as to methods for reducing the incidence of these tragedies, and is derived from a critical review of the records.

There is a general feeling among surgeons that the incidence of cardiac arrest is on the increase. There is some feeling, too, that the term cardiac arrest has been widely misused in hospitals. It has been recently suggested that there be accurate registration of deaths occurring during anesthesia and surgery by a central agency.[2]

Cardiac arrest has been described as an unexpected, rapid development of asystole or ventricular fibrillation which is potentially avoidable and potentially reversible. Collins[5] has recently suggested that the term "cardiac arrest" be discarded, and that the term "cardiocirculatory collapse" be used.

Cardiac arrest may occur under what may be considered ideal circumstances; however, in many instances there may be a warning sign. The accumulated knowledge of the causes of cardiac arrest is now considerable and may be complicated.

All authorities are agreed that if successful resuscitation without cerebral damage is to be accomplished, it must be done in a period of not more than 3 to 5 minutes. This is truly an operating room emergency (table 1).

MANAGEMENT OF PATIENTS

It is not within the scope of this paper to attempt to detail a plan for the prevention and treatment of cardiac arrest. Thorough and careful consideration of these details has been outlined by Hosler,[8] Beck and Leighninger[1] and others.

Certainly, there must be good liaison between the surgeon and anesthetist. The proper evaluation of the operative patient, particularly in regard to preoperative medication and proper fluid and electrolyte balance, is mandatory. It is the opinion of the authors that every patient who is taken to surgery should be monitored during the entire operative procedure.

It should be the concern of the surgical staff, in cooperation with the anesthesia staff and the hospital administration, to see that the entire hospital staff (attending, house, nursing, and others) is alerted to the possibility of cardiac arrest.

When the anesthesiologist has announced to the surgeon that cardiac arrest has occurred, diagnosis should be confirmed within 15 to 20 seconds, the surgeon should be prepared to pick up the scapel, and start massaging the heart within another 30 to 60 seconds. Each hospital should have these details well planned in advance. The total management of this condition may be complicated, but fundamentally the immediate problem is to deliver oxygen to the brain, myocardium and other vital tissues. The major cause of death after successful cardiac resuscitation is injury to the central nervous system. One of the most encouraging recent recommendations for treatment of this grave condition concerns the use of hypothermia. Rosomoff[9] and others have investigated the protective value of hypothermia in states of cerebral ischemia. Williams and Spencer[11] of Johns Hopkins Hospital reported 4 patients who exhibited severe neurologic injury after successful cardiac resuscitation. All 4 patients reported by this group had undergone cardiac massage and exhibited severe neurologic injury. They were treated promptly with hypothermia; 3 patients recovered completely, and the residual neurologic defect in the fourth is of moderate severity.

SELECTION OF PATIENTS

We have eliminated from this study the operating room deaths which obviously were not

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cardiac arrest cases. In some instances it is difficult to decide to which category certain cases should be assigned.

There is some confusion as to just what constitutes and justifies the diagnosis of cardiac arrest. Some of the patient charts were not signed out with this diagnosis. Obviously, in such instances it is difficult to search out such cases for statistical review.

Our methods for tracking down cases were as follows: (1) All operating room deaths listed by the operating room supervisor were studied. (2) The record room index was helpful in locating those cases which had been signed out as cardiac arrest. (3) Cards were sent to the entire hospital attending staff, requesting that they contact the authors to give them pertinent and appropriate information on any case with which they might be familiar. (4) Finally, at a called meeting of the house staff, the residents and interns were asked to recall any cases in their own personal experience. In this manner we feel that very few, if any, cases have escaped our attention. We wish to emphasize that if cardiac arrest has occurred, it should be entered as such into the hospital record. It is only in this manner that reliable information regarding these cases may be obtained.

TABLE 1
Time elapsed before cardiac massage

Cardiac Arrest	No. of Patients	Recovered	Died	Per Cent Recovered
Less than 5 min.	16	6*	10	38
More than 5 min.	4	0	4	0

* One patient with severe neurologic residual.

TABLE 2
Age of patients with cardiac arrest

Decade	No. of Patients
years	
0–9	7
10–19	2
20–29	1
30–39	1
40–49	5
50–59	4
60–69	3
70–79	2
80+	2

TABLE 3
Cardiac arrest cases

Classification	Distribution
Service	8
Private	19
Total	27

TABLE 4
Type of surgery with cardiac arrest

Type of Surgery	No. of Patients
Neurosurgery	2
Abdominal	11
Genitourinary	2
Thoracic	5
Head and neck	1
Orthopedic	2
Eye, ear, nose and throat	4
Total	27

TABLE 5
Preoperative physical status of 27 patients with cardiac arrest

Condition	No. of Patients
Good	6
Fair	10
Poor	4
Serious	4
Emergency—good	0
Emergency—poor	1
Moribund	2

INCIDENCE OF CARDIAC ARREST

It is felt that periodic careful appraisal of all operating room deaths is indicated in both teaching and private hospitals.

This report constitutes a study of all cases of cardiac arrest occurring in St. Anthony Hospital, Oklahoma City, Oklahoma, during the period January 1, 1953, to January 1, 1960. St. Anthony Hospital is a large general hospital admitting over 20,000 patients per year. It has the advantage of a large intern-resident staff, and is associated with the teaching program at the University of Oklahoma School of Medicine.

Approximately 20 per cent of the cases at the hospital are service cases and are admitted to the teaching service.

The age distribution by decade is shown in table 2. The relative distribution of occurrence in private and service cases is shown in table 3. House staff indoctrination on cardiac arrest (by staff conference) is included in the educational program at this hospital during the first week in July of each year.

The operation or proposed operation in each case of cardiac arrest ranged in severity from simple cataract extraction with local anesthesia to thoracotomy (table 4).

PHYSICAL STATUS

The cases have been classified as to physical status according to the recommendations of the American Society of Anesthesiologists (table 5). It is interesting to note that only 6 of the 27 cases, or 22 per cent, were judged as "good." In some instances, the "fair" risks could very well have been included in the "poor" category.

Some of the most striking and unusual of our cases concern cardiac arrest which occur during ophthalmic procedures. Two of the patients reported had a fatal outcome. Another apparently incipient "near cardiac arrest" case is reported here in some detail.

It is well known that pressure on the eyeball may produce bradycardia. It is not generally appreciated that cardiac arrest may occur as a result of ocular pressure or pull on an extraocular muscle during ophthalmic surgical procedures.

There have been 2 such patients[6] observed in the past 2 years, both under local anesthesia, in preparation for cataract extraction. One, F. M., a white 70-year-old woman, will be described here. The cataract had been extracted successfully from her right eye the week before, and she was being prepared for operation upon the left eye. The same technique was employed. She was in good health and alert; the blood pressure was 135/70 15 minutes before the near-fatal episode. There were 3 to 4 cc. of 2 per cent Xylocaine injected to produce lid akinesia, and 2.0 cc. were injected behind the globe in the orbit. To spread the anesthetic agent and to soften the globe, pressure was made on the eyeball through the closed lid. The patient chatted freely with the surgeon during this procedure, but after 3 min. of ocular pressure ceased talking. Upon removal of the head drapes, she was found to be unconscious, with respirations of 6 to 8 per min.; blood pressure, 60/0; and heart rate, 40. Oxygen was administered with a closed system. The blood pressure and pulse remained unchanged for several minutes and intravenous atropine, $\frac{1}{120}$ gr., was given. Within less than a minute the pulse rate increased to 60 and the blood pressure began to rise. She improved steadily, became conscious within minutes, and made a complete recovery.

The mechanism involved is the oculocardiac reflex, which travels *via* the sympathetic fibers in the orbit over the branches of the trigeminal and thence to the vagus nerve. In the case just described, excellent evidence that the oculocardiac reflex was acting was furnished by the immediate response to intravenous atropine. This drug used *intravenously* is *specifically* for bradycardia due to ocular pressure. If used intramuscularly, it is of no value in this condition.

TABLE 6
Anesthesia used in cardiac arrest cases

Anesthesia	No. of Patients
Barbituate (intravenous)	
with muscle relaxant and nitrous oxide-oxygen	11
with local anesthesia	1
with muscle relaxant and nitrous oxide-oxygen and cyclopropane	1
with oxygen	1
with nitrous oxide-oxygen	1
Spinal anesthesia	2
Ether-oxygen	1
Vinethane and ether	4
Cyclopropane and ether-oxygen	1
Open drop ether with local Novocain	1
Local Xylocaine	1
Topical Nupercaine	1
No anesthetic*	1

* Barbituate poisoning in 22½-month-old child.

ANESTHESIA

The anesthetic agents used appear to have no correlation to the incidence of arrest. Cardiac arrest occurred with various anesthetics as shown in table 6. It would be difficult, indeed,

TABLE 7
Time at which cardiac arrest occurred

Time of Arrest	No. of Patients
Before surgery began	7
During surgical procedure	13
Surgical procedure concluded	7
Total	27

TABLE 8
Incidence of cardiac arrest

Incidence	No. of Patients	Per Cent
Total surgical procedures (major and minor) 1/1/53 to 1/1/60	63,307	
Total no. of cardiac arrest cases	27	0.042
Deaths due to cardiac arrest	20	0.031
Survival after cardiac arrest	7	

to indict the so-called "cocktail anesthesia," from a study of our records; similarly, no indictment could be made against any singular preanesthetic medication.

The most dangerous periods of anesthesia are usually stated to be on induction and during emergence. We could detect no most dangerous period in this study (table 7). Obviously, cardiac arrest can be just as deadly after a long technically successful operative procedure (while the bandage is being applied) as during induction or any other stage.

RESULTS OF TREATMENT OF CARDIAC ARREST

Table 1 points out the necessity for haste in performing cardiac massage. Table 8 shows that this catastrophe occurred 27 times in 63,307 total surgical procedures, for an incidence of 1 in every 2344 cases.

COMMENT

While our series of cases may be too small to be statistically significant, certain facts were brought to mind by a study of records and careful consideration of the etiology and management of this condition. (1) Arrest may occur at any time as the result of acute anoxia, chronic hypoxia or acute hypoxia superimposed on a chronic hypoxic state. (2) It is realized that the cause or causes of cardiac arrest may be many and complicated; however, we feel that the following points are worthy of extra consideration and emphasis, for they may contribute to the production of cardiac arrest: (a) even a moderate overdose of anesthetic agent, (b) respiratory depression caused by anesthetic agents not compensated by adequate assisted or controlled respiration, (c) prone position with no control of airway or provision for artificial respiration, (d) respiratory obstruction by position of the head, dislodgement of endotracheal tube, kinked endotracheal tube, an endotracheal tube which was too long and produced massive atelectasis, (e) lack of recognition of hypoxia state, (f) hypoxia and hypercarbia induced by open drop ether mask, (g) respiratory depression and/or obstruction following removal of mask at end of operation not noted, (h) profound high spinal anesthesia with respiratory and circulatory depression not recognized or compensated for, (i) inadequate evaluation and treatment of patient preoperatively, (j) long continued respiratory obstruction resulting in pulmonary edema, and (k) operating time unduly prolonged.

SUMMARY

We have presented our analysis of all cases of cardiac arrest which have occurred in St. Anthony Hospital, Oklahoma City, Oklahoma during the period January 1, 1953, to January 1, 1960.

It is realized that such a series in a single general hospital is not statistically significant; however, it is our opinion that regular periodic review of these cases should be carried out and presented. Thus the entire hospital staff will become more cognizant of a condition which, at least in some instances, may be preventable and in others, where it has been allowed to occur, may be reversible.

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LEIOMYOSARCOMA OF THE STOMACH (EXOGASTRIC): CASE REPORT

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Leiomyosarcomas[1, 2, 3, 5] are rare, especially in the stomach. On x-ray they may be present as ulcer-like lesions or as large, cystic tumor masses of the abdomen. The surgeon, while exploring the abdomen for a questionable liver or spleen lesion, is surprised when he finds this large stomach tumor. We present a case of a large cystic mass of the abdomen, a leiomyosarcoma, as another diagnostic problem of cystic tumors of the abdomen.

CASE REPORT

This 67-year-old white man, a retired miner, was first seen at the Ohio State University Hospital with the history of an abdominal mass, present for 3 weeks, but growing progressively larger. He had noted, on occasion, in the last 6 months, a fullness in the epigastrium and some vague discomfort in the upper abdomen on eating. A week before admission, a white blood count was 59,000 with 90 per cent lymphocytes.

On examination of the abdomen, a large, movable, 10 by 12 cm. cystic mass was found, which was in the midepigastric to the right lower quadrant position. The mass was nontender. The remainder of the physical examination was normal.

The laboratory data again showed the white blood cell count to be 59,000 with 92 per cent lymphocytes. Liver studies and blood urea nitrogen were normal. A bone marrow study showed moderate infiltration with lymphocytes.

It was felt this was a mesenteric cyst, and on February 4, 1958, under general anesthesia, an exploration was performed. A large cystic mass was found. It was closely bound to the stomach and the transverse colon. It was mobilized down to the gastric wall where it was found to be continuous with it. It was removed without difficulty. The postoperative course was satisfactory and the patient was discharged from the hospital on the twelfth postoperative day.

DISCUSSION

Leiomyosarcomas are rare. One of every 100 malignant lesions of the stomach is a sarcoma and one of every 10 of these is a leiomyosarcoma.[2, 6, 9] The leiomyosarcomas are of 2 types, submucosal (endogastric) or subserosal (exogastric). Of these, only about 10 per cent are exogastric.

In 1762, Morgagni[2, 7] first described the gross pathology of gastric sarcoma. Bruch, in 1847, gave the first case report of gastric sarcoma, and in 1863, Virchow[7] reported the first myosarcoma of the stomach. Knowledge of this tumor has been slow and only since 1940 have the number of cases increased notably.

On microscopic examination the lesion shows spindle-shaped cells with elongated nuclei and fibrillar stroma. The origin of this tumor is questionable. Ewing[4] feels the tumor is malignant at the start, but Conheim[7] suggests that these are embryonal cell arrests.

The tumor metastasizes slowly and differentiation is late. Thus, the patient's prognosis is usually good after the tumor is removed. Marvin and Walters[8] reported a case of 20-year survival postoperatively.

Although the preoperative diagnostic percentage is low (10 per cent), careful and thorough removal of the tumor has given good survival. In our own case, after the lesion was identified and carefully removed from the stomach wall, we felt the prognosis to be good. A 2-year follow-up shows the patient to be in good health with no evidence of tumor recurrence.

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A PHOTOGRAPHIC ORIENTATION OF THE PANCREATIC DUCT ORIFICE

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A reviving interest in pancreatic disease, especially pancreatitis and its sequellae, will undoubtedly cause surgical attention to be focused upon the pancreatic ductal system. Since the best surgical approach to the pancreatic ductal system is through the papilla of Vater, it is imperative that the location, relationships and variations of the pancreatic duct orifice be understood. Bartlett and Nardi[1] have illustrated these features in line drawings, and Coffey and Bollinger[2] have pointed them out in their motion picture describing the technique for pancreaticolithotomy. It has been our experience, however, that locating the pancreatic duct orifice requires a degree of patience and familiarity which we have found can be gained in dissecting the fresh autopsy specimen of duodenum, common bile duct and pancreas.

We have recorded the dissections of 25 fresh autopsy specimens‡ on type F Kodachrome film through the use of an Exacta camera equipped with a 13.5-cm. telephoto lens which was capped with an additional portrait lens. The first four dissections were a dismal failure because we attempted to locate the pancreatic duct orifice through the dilated sphincter without performing a sphincterotomy.[2] In one preparation of this group we were successful because the pancreatic duct orifice was completely separate and distinct from the papilla of Vater. This situation is shown in figure 1.

It became apparent to us that a technique would have to be devised which would be applicable in the operating room, and which would facilitate a consistent and rapid visualization of the pancreatic duct orifice. This, we feel, we have simply and successfully accomplished by what we have chosen to call the triangulation of the sphincterotomy incision as demonstrated in figure 2. A probe is inserted into the common bile duct through the papilla of Vater and an incision is made over the probe through the posterior wall of the duodenum and the anterior wall of the common bile duct for a distance of about 1.5 cm. The probe is then lifted into the incision and bent so that the apex of the incision becomes a fixed point against the probe. Silk sutures, placed through the mucosa of the duodenum and common bile duct at the rim of the papilla of Vater on either side of the sphincterotomy incision, are pulled taut in a lateral direction. These maneuvers form a triangle which exposes the posterior wall of the common bile duct and the posterior edge of the papilla of Vater. It is in this area, which can be minutely examined, that the orifice of the pancreatic duct will be found most commonly.

In the 25 preparations dissected, we found that in 3 instances the pancreatic duct orifice was located on the edge of the papilla. In 16 instances it was just inside the papilla. In 2 preparations it was located a considerable distance away from the papilla but within the common bile duct. In 4 cases we were unable to be specific because the dissection made the landmarks indistinct. (These preparations were attempted without the benefit of triangulation.) An accessory duct was found in 4 cases and in 1 of these it represented the only pancreatic duct. Figure 3 is an illustration of this situation.

In each of 4 cases, a duodenal diverticulum was found to be located in such close association with the distal portion of the common bile duct and the papilla of Vater that the risk of diverticulectomy would have been exceedingly great. Figure 4 is an illustration of this situation.

The usually close relationship of the pancreatic duct and its orifice to the common bile duct and the papilla of Vater is well illustrated in figures 5, 6, and 7.

Pancreatography is a promising tool for the study of pancreatic disease. Pancreatograms, done either in the operating room or at the dissecting table, do not ordinarily require a large amount of opaque medium. Figure 8 is an illustration of a pancreatogram of a freshly dissected specimen. There were 3 cc. of Hypaque

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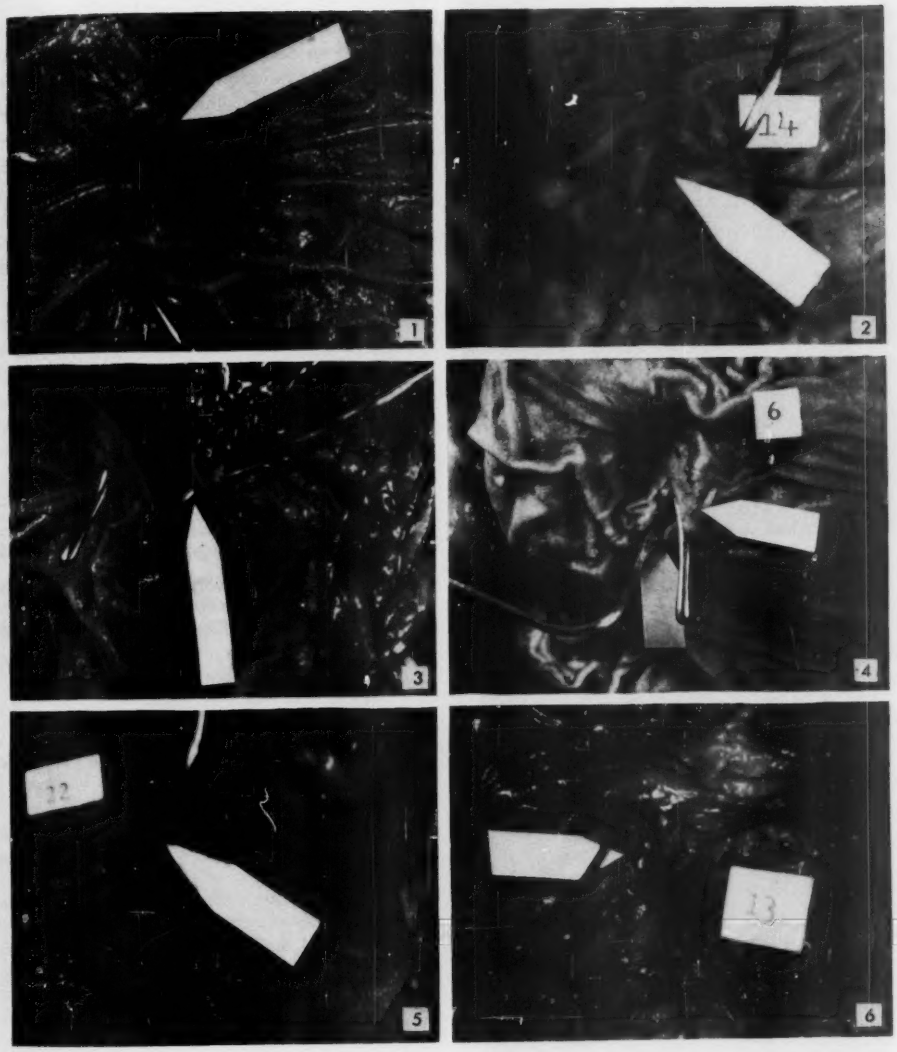
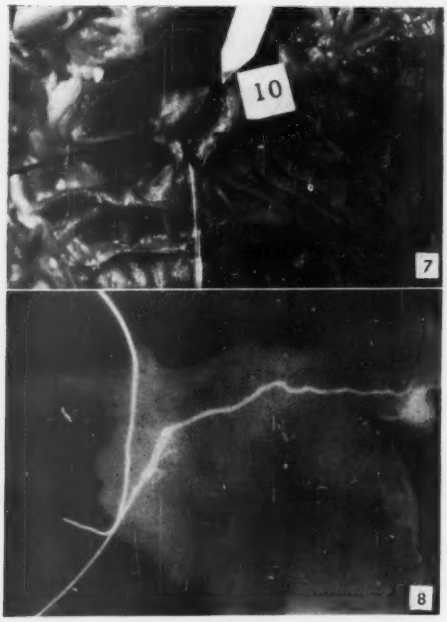


FIGS. 1 TO 6

FIG. 1. Pancreatic duct enters duodenum apart from papilla of Vater. FIG. 2. The triangulation of the sphincterotomy incision. FIG. 3. Main duct as accessory duct. FIG. 4. Duodenal diverticulum in close relationship to common bile duct and the papilla of Vater. FIG. 5. Pancreatic duct orifice on posterior rim of papilla. FIG. 6. Pancreatic duct bends to the right as the common bile duct ascends behind the duodenum.



FIGS. 7 AND 8

FIG. 7. Pancreatic duct orifice inside rim of papilla. FIG. 8. Pancreatogram of dissected specimen.

used, which can be seen to fill the duct and escape from the distal end of the duct where the pancreas was cut away from the spleen.

By dissecting 25 autopsy specimens, we have developed an easy, consistent and rapid method to locate and intubate the pancreatic duct orifice. We feel that this technique will be useful for pancreatography, pancreaticolithotomy, and for experimentally measuring pancreatic duct pressures. The ease of locating the pancreatic duct orifice by triangulating the sphincterotomy incision has made us hopeful that advances can be made in understanding the enigma of the diseased pancreas.

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OPERATIVE TREATMENT OF THE SUPERIOR MESENTERIC ARTERY SYNDROME

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The clinical entity of obstruction of the duodenum at the site of its passing under the superior mesenteric artery has been described as being acute, chronic or intermittent in type.[2, 3] It has many eponyms, among them being duodenal ileus (Wilkie's syndrome), acute gastroduodenal obstruction (dilation), arteriomesenteric duodenal compression and superior mesenteric syndrome.[6] Many cases have been reported in the literature; and extensive publication of articles relating to the signs, symptoms and treatment of the disease have been recorded throughout the past 50 years. In a recent publication, Kaiser, McKain and Shumacker[4] described their experiences with 18 patients treated surgically over a period of 6 years and defined the 3 types of obstruction and related their experiences in the treatment. It is the purpose of this article to give a description and amplification of the surgical treatment. An illustrative case is presented.

CASE REPORT

Mr. F. B., a 35-year-old white man, was admitted to the hospital on September 17, 1959, with a history of upper abdominal pain and distress of about 2 months' duration, and nausea and vomiting for 24 hours. The tentative diagnosis was acute peptic ulcer.

He gave a history of having had a similar episode of this severity approximately 8 years previously, at which time he was hospitalized and a gastrointestinal series was made (fig. 1). This gastrointestinal series was erroneously interpreted as being essentially normal. He continued to have similar bouts of nausea, vomiting and upper abdominal distress intermittently and he noted that he was unable to maintain what he considered a normal weight throughout this 8-year period of time. He stated that he had a sensation of hunger most of the time; but that eating caused nausea and an excessive amount of food resulted in vomiting or a sensation of fullness in the upper abdomen. He had taken antacids and antispasmodics, but obtained no relief of his symptoms. Throughout this 8-year period he maintained a diet of relatively soft food taken in frequent, small amounts.

Examination revealed a 35-year-old white man, appearing chronically ill, thin and undernourished. He was 5 feet 10 inches tall and weighed 129 pounds. His blood pressure was 110/68, pulse 80 and temperature 99. His white blood count was 8100 with a normal differential, and his hemoglobin level was 13.8 gm. The heart and lungs were negative. Examination of the abdomen revealed tympany and distention of the upper abdomen and peristaltic sounds were hypoactive. There was mild tenderness of the epigastrium and right upper quadrant.

Upon admission a nasogastric tube was inserted; gastric contents were aspirated and the patient obtained great relief. The following day an upper gastrointestinal series was made (fig. 2). The

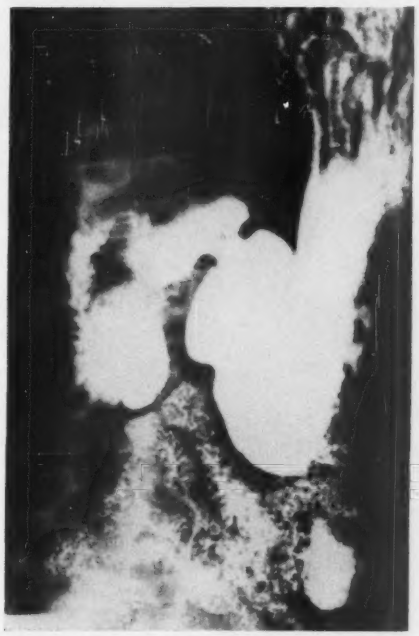

FIG. 1. May 12, 1953. Original examination. Dilation of horizontal duodenum. Regarded as normal by radiologist.

gastrointestinal series revealed obstruction of the third portion of the duodenum and compression by the superior mesenteric artery. After reviewing old films and determining the chronicity of the patient's symptoms, we advised surgical exploration. Preoperative preparation was carried out in the form of parenteral fluid, electrolyte and vitamin therapy. On September 25, 1959, surgical exploration was done. The ligament of Treitz was severed and the duodenojejunal juncture was mobilized in the manner to be described in the following section. The patient made an uneventful recovery and began taking oral fluids on the second postoperative day. He was tolerating a regular diet on the third postoperative day and on the fifth postoperative day he stated that he could eat "better than he had for years." His convalescence was uneventful and, 10 months following surgery, his weight was 160 pounds, a gain of 30 pounds. He has no upper gastrointestinal symptoms whatever, and an upper gastrointestinal series made 3 months postoperatively is shown in figure 3.

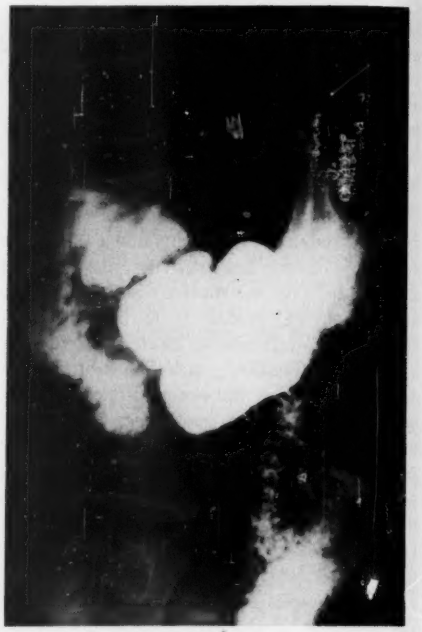

FIG. 3. January 14, 1960. Postoperative film shows normal sized stomach and duodenum. Trituration of mild degree persists.

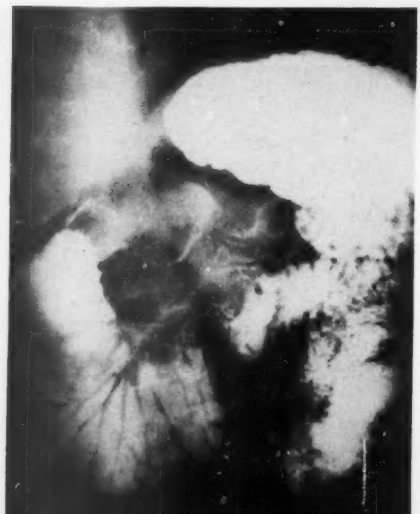

FIG. 2. September 18, 1959. Massive dilation of duodenum and stomach, and marked trituration; slightly oblique shadow crossing distal duodenum actually represents the superior mesenteric artery.

OPERATIVE PROCEDURE

The operative technique employed in the foregoing case is described in figures 4, 5 and 6.

An upper abdominal midline incision or left paramedian incision may be employed and exploration of all abdominal viscera should be carried out initially. The stomach will usually be found to be considerably dilated; the first and second portions of the duodenum can be seen to be enormously dilated, often containing a great deal of air, and the wall is hypertrophic. This may usually be noted in spite of an indwelling Levin tube in the stomach. The greater omentum and transverse colon are then retracted superiorly, and the site of angulation of the duodenojejunal flexure should be noted (fig. 4). An attempt to introduce a finger adjacent to the duodenum at the very site where the superior mesenteric crosses the duodenum will reveal the duodenum to be very tightly bound in this angle. The superior mesenteric artery has the characteristic

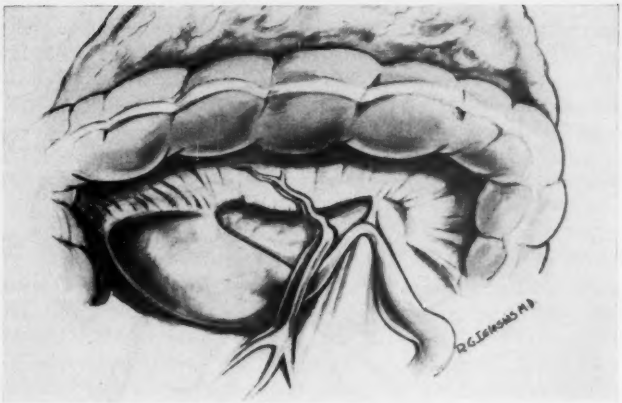

FIG. 4. Obstruction of duodenum beneath superior mesenteric artery

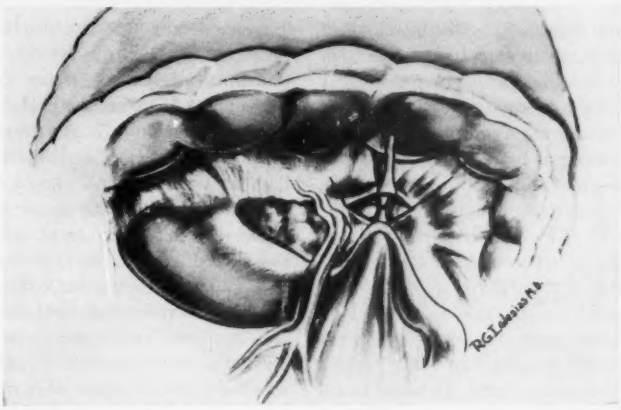

FIG. 5. Incised peritoneum and partially severed ligament of Treitz

of a firm cord or band across the duodenum. This is the point of obstruction. The duodenojejunal flexure is very tensely anchored in a high position by the ligament of Treitz, which virtually pulls the third portion of the duodenum into this acute vascular angle, causing the fourth portion of the duodenum to assume a vertical position (fig. 4).

Step 1. The peritoneum overlying the ligament of Treitz and the paraduodenal fossae should be incised transversely (fig. 5). This incision should be approximately 3 to 4 inches in length. Upon retraction of the peritoneum and by blunt dissection through the underlying loose areolar connective tissue, the ligament of Treitz is readily visualized. The inferior mesenteric vein and the aorta may be visualized to the left of the ligament of Treitz.

Step 2. Figure 5 illustrates the peritoneum incised transversely and the ligament of Treitz partially severed. Paradoxically, the ligament of Treitz is more of a muscular band than a ligamentous band. The so-called ligament or muscle of Treitz is a smooth muscle bundle which is usually a flat, broad, muscular band approximately 1½ to 2 cm. in width and tapered. When this smooth muscle has been severed transversely, it will be noted that it has a rich blood supply and several small blood vessels need to be ligated. It may also be noted that there are several small duodenal veins which

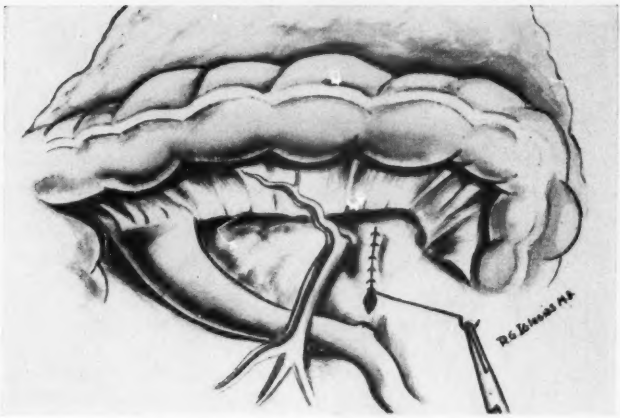

FIG. 6. Longitudinal closure of peritoneum

should be ligated adjacent to the ligament of Treitz. When the duodenojejunal angle has been liberated, the duodenum will drop remarkably away from the vascular angle and two or three fingers may be placed adjacent to, and parallel with, the third portion of the duodenum beneath the superior mesenteric vessels to verify the adequacy of the duodenal channel at this point.

Step 3. (Fig. 6). The fourth portion of the duodenum will be noted to distend and assume a more normal size and position after it has been mobilized. By applying traction downward on the duodenojejunal angle, the transverse peritoneal incision is now converted to a longitudinal one, and the peritoneum is closed in a longitudinal manner. This has a two-fold purpose. First, it displaces the duodenojejunal flexure downward and maintains it in its newly created position. Second, it closes the paraduodenal fossae and the posterior parietal peritoneum and duodenal fossae to prevent paraduodenal herniation. The fourth portion of the duodenum and the proximal jejunum will immediately distend and so give evidence of free passage of duodenal contents beneath the superior mesenteric artery.

This procedure has been found to be a great simplification of the operative treatment of this condition and eliminates the need for duodenojejunostomy and all the dangers attendant to the performing of an anastomosis.

DISCUSSION

The principle of the division of the ligament of Treitz for arteriomesenteric obstruction was first proposed by Strong[5] in 1958. He proposed that the etiology of this type of obstruction was the maintenance of the duodenum in the acute angle between the aorta and the superior mesenteric artery by abnormal fixation of the ligament of Treitz; and that this abnormally high fixation resulted in acute angulation of the duodenum in this vascular angle. He presented a case whereby the ligament of Treitz was divided and the duodenum was mobilized; a dramatic cure of his patient ensued. The etiology of this syndrome remains unsettled. Some of the theories proposed are: increased lumbar lordosis, abnormally heavy mesentery and intestine, redundancy of the cecum, abnormal fixation of the ligament of Treitz and excessively acute angulation between the superior mesenteric artery and the aorta. However, none of these foregoing theories adequately explains why most of these cases occur in the third and fourth decades of life, the highest incidence between the ages of 25 and 35.

Most textbooks of anatomy published within the past three decades are in agreement that the ligament of Treitz is not in reality a ligament, but a muscle. Batson[1] and others have long ago drawn attention to this anatomic misnomer and misconception as to the integrity of the structure. It is a well defined band of smooth muscle which cannot be construed as a ligament. It is our belief that this high fixation of the duodenojejunal angle is due to hypertrophy and excessive contraction of this muscle which results in an abnormally high fixation of the duodenojejunal

flexure. The abnormal contraction and fixation and hypertrophy of this smooth muscle could be likened to hypertrophic pyloric stenosis or to torticollis. This theory of hypertrophy would indeed account for the acquired nature of the condition as well as its intermittency. Further histologic study is necessary to lend credence to this theory.

The simple division of the muscular ligament of Treitz and the longitudinal closure of the parietal peritoneum have simplified the treatment immeasureably and do not alter the continuity of the intestinal tract. Duodenojejunostomy and gastrojejunostomy have been employed for the past 30 years for the relief of the obstruction by bypass procedure. These bypass procedures did effect a cure, but these are ofttimes technically difficult, especially a duodenojejunostomy. There are late complications of bypass procedures, namely, marginal ulcers following gastrojejunostomy and malabsorption syndrome, and anemia following duodenojejunostomy. The duodenal mobilization procedure is, of course, not attended by these complications and does not disturb the normal physiology of the alimentary tract.

The question arises as to whether there is any disability or alteration of the physiology resulting from the division of the ligament of Treitz and from the loss of this suspensory ligament of the intestine. The main suspension of the small intestine when the individual is in an erect position would lie entirely upon the mesentery and the superior mesenteric artery and vein. One would expect the patient in the postprandial period, after assuming a vertical position, to experience abdominal pain or autonomic nervous system reflexes due to traction on the root of the mesentery by the distended small intestine. In the 10 months postoperatively, we have followed this patient for postprandial cramping abdominal pain or symptoms of a "dumping syndrome." He has remained completely asymptomatic. One cannot conclude from this meager experience that the muscular ligament of Treitz is completely a dispensable structure, but sacrifice of the structure apparently does not result in disability.

SUMMARY

1. Reference is made to the many names and clinical descriptions of superior mesenteric artery obstruction of the duodenum.

2. An account of a simplified operative procedure for the treatment of superior mesenteric artery syndrome by transection of the ligament of Treitz and inferior displacement of the duodenojejunal flexure is presented.

3. The relative merits of duodenal mobilization as contrasted to anastomotic bypass procedures are summarized.

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THE SIGN OF INDIRECT INGUINAL HERNIA*

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The most commonly quoted differentiating features of direct and indirect inguinal hernia are shown in table 1. These points are useful clues, but they are not evidence. Their use is a devious approach to a presumptive distinction. The sum of them does not add up to a positive diagnosis. An asthenic, retired Colonel who complains of bilateral, easily reducible groin swellings after toting rocks to build a patio and barbecue pit may be considered to have direct hernias simply on the basis of probability. But even if examination shows large external rings and flaccidity in Hesselbach's triangle, there is no assurance that he does not have an indirect hernia. Often he does, but probability will not make a diagnosis.

TABLE 1

Differentiating features of direct and indirect inguinal hernia

Features	Indirect	Direct
Age	Younger	Older
Abdominal wall	Normal	Weak
External ring	Not necessarily enlarged until late stage of large hernia	Always enlarged
Position	Oblique in groin; frequently scrotal	Localized near external ring; seldom scrotal
Shape	Ovoid, pear-shaped, or fusiform	Dome-shaped, or globular
Bilateral?	About one-quarter of cases	Over one-half of cases
Inguinal canal	Posterior wall firm	Posterior wall weak
Relation to cord	In front or on outer side	Above and to inner side
History	? Congenital	Strain; chronic or recurrent increase in intra-abdominal pressure

A straightforward approach to differentiation between direct and indirect hernia is usually as simple as their names. The direct hernia passes straight (directly) through the abdominal wall to the external ring. The indirect hernia approaches the external ring (indirectly) by way of the internal ring and the inguinal canal.

Examination of hernia customarily centers about the external ring. If the abdominal wall offers little resistance to an examining finger which is pushed along the axis of a direct passage, and if several of the secondary clues are present, the examiner feels justified in making a diagnosis of direct hernia. But neither the easy passage, nor the age of the patient, nor the size, shape, and location of the swelling warrants the conclusion that the sac of the hernia of which the patient complains has come through the hole that the finger is poking. The patient may have both a direct and an indirect hernia. He may not have a direct hernia at all. A thin posterior wall that yields readily to the tip of a finger may hold back the abdominal contents with only a slight general bulge. If the "direct" repair in such a case does not include incision of the cremaster and careful exploration of the cord, the sac of the indirect hernia will be missed, and the end result of the repair will be somewhat less than optimum.

The external ring is one point in human geography that direct and indirect hernias share in common. The key to distinction between the two types is examination of the internal ring. If it can be shown that a hernia passes through the internal ring, it is certainly an indirect hernia. If it cannot be demonstrated that the hernia does pass through the internal ring, the hernia is most likely direct.

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In a patient who is not obese, the internal ring can sometimes be palpated. Rarely, its conformation and exact location is even visible through a thin abdominal wall. But even when the ring itself cannot be felt, the relation of the hernia to it can usually be determined if the hernia is reducible.

The maneuver (figs. 1 to 5) is: (1) reduce the

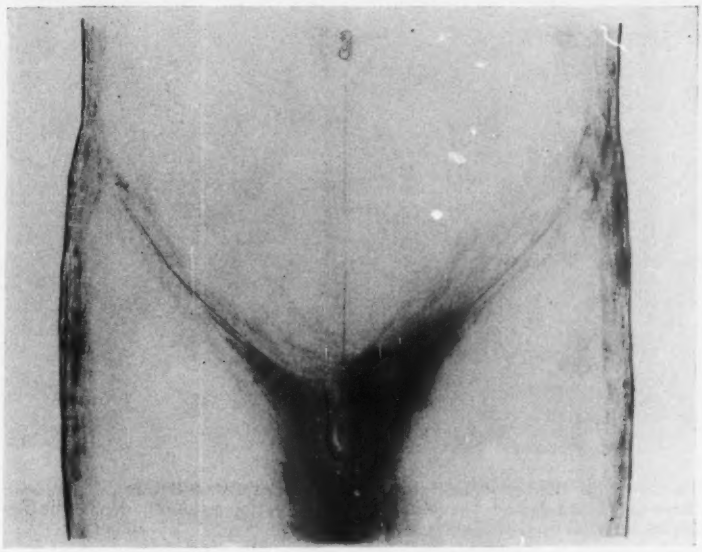

FIG. 1. Presumptive direct hernia on left

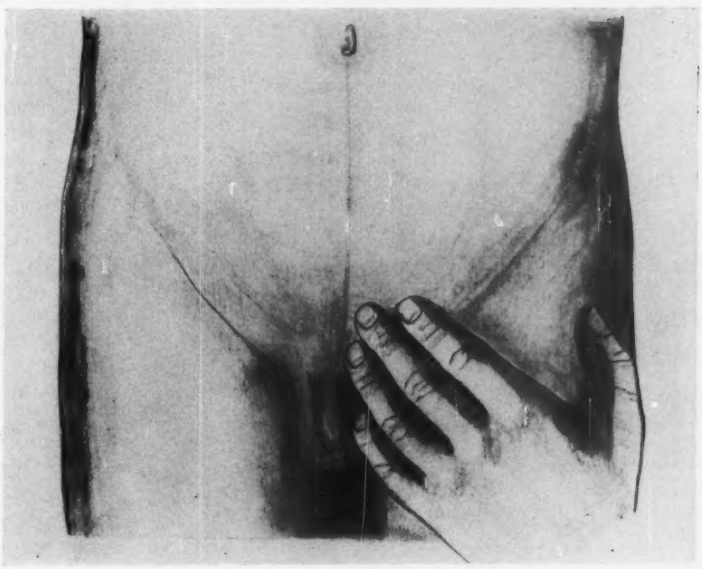

FIG. 2. The hernia on the left is reduced by the pressure of the last three fingers of the surgeon's left hand.

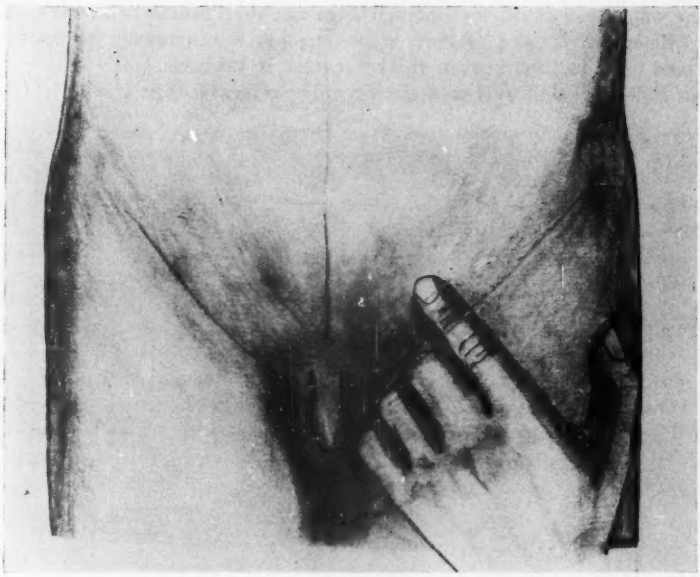

FIG. 3. After reduction, the tip of the index finger is placed over the internal ring, and the pressure of the other three fingers is released; the hernia does not recur on straining, demonstrating that the sac passes through the internal ring.

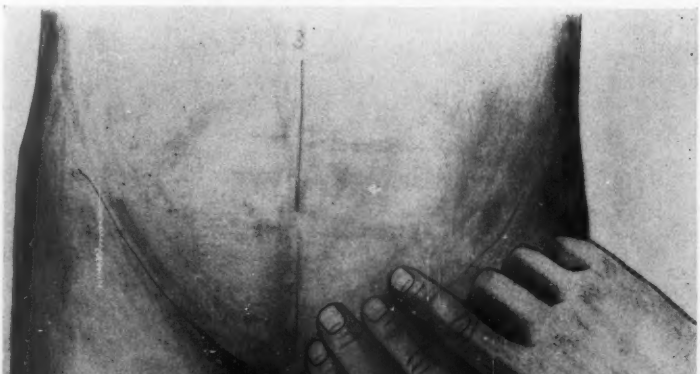

FIG. 4. If the hernia is large, and the anatomy is distorted, four fingers are used in reduction

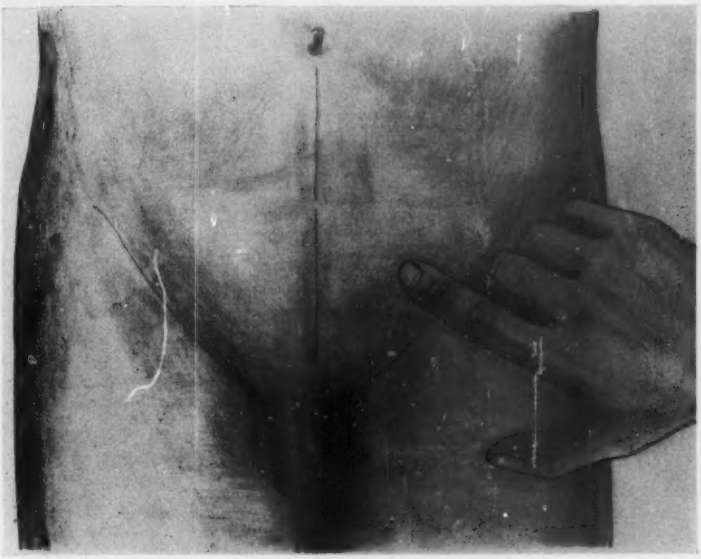

FIG. 5. The index finger remains flat across the lateral border of the hernia when the pressure of the other fingers is released.

hernia; (2) occlude the internal ring; (3) release the reducing pressure and observe; and (4) release the occluding pressure and observe.

The location of the internal ring can be estimated as just above the inguinal ligament, about halfway between the pubic tubercle and the anterior superior iliac spine. Pinpoint accuracy in placing of the tip of a finger is not essential; a finger across the lateral portion of the canal will do as well. If the opening of the hernial sac is large, in either direct or indirect hernia, the anatomy is distorted; to dependably "occlude the internal ring" (step 2) requires placing a finger or thumb across the lateral edge of the collapsed hernial sac, where the internal ring "ought" to be. This will not interfere with the validity of the test. It will not completely occlude the neck of a direct hernia, and will usually occlude the neck of all but the largest indirect hernias.

If the hernial protrusion recurs medial to the occluding finger while the internal ring is properly occluded, there is a direct hernia. If finger pressure over the internal ring keeps the hernia reduced, the hernia is indirect; its course can be observed as finger pressure is released. Whether the patient is husky or senile, or has one hernia or two, is immaterial. The difference between a direct and an indirect hernia is which way it gets through the fence, not what it looks like on the other side.

This is not a new or original observation. It has been known for years. Occasionally it rates a passing sentence in a current text. But so often has its demonstration to a colleague been accepted as a novel idea that it appears warranted to publicize the maneuver. Examination of the internal ring is a simple, logical, direct means of determining preoperatively the presence of an indirect hernial sac.

Acknowledgment: I am grateful to Private First Class Paul L. Paquette for the illustrations.

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GASTRIC HEMORRHAGE DUE TO A SUBMUCOSAL LIPOMA*

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Submucosal lipoma of the stomach, even though encountered relatively infrequently, is clinically important. Failure to recognize such a lipoma and to remove it may result in exsanguinating hemorrhage.[9]

Sporadically, case reports on a symptomatic gastric lipoma have appeared in the surgical literature.[1-5, 9] The rarity of this lesion is emphasized by Stout's data.[7] He found that, in 651 benign and malignant gastric tumors which were examined at the Columbia University Laboratory of Surgical Pathology during a 42-year period, only one lipoma was found, and no liposarcomas were encountered. The incidence in this series of gastric tumors was 0.2 per cent of all gastric tumors or 0.9 per cent of benign stomach tumors.

The following case is reported to emphasize that a stomach lesion, which is associated with severe hemorrhage and which has possibly the appearance of a carcinoma with radiographic studies, may be an easily resectable and curable lipoma.

CASE REPORT

Chief complaint. Tarry stools for 5 days.

Present illness. On May 24, 1960, this 62-year-old white woman said that she noticed tarry stools about May 19. She did not vomit any blood and she did not have abdominal pain. She noticed that, in the week before admission, she had become progressively weaker.

Physical examination. General surgical examination was not remarkable except for a marked paleness of the skin. No abnormal abdominal masses were palpable.

Laboratory data. At the time of admission, the hemoglobin was 6.3 gm. per cent, and the hematocrit was 20 vol. per cent. The total blood volume was 4020 cc.; the plasma volume, 3300 cc.; and the red cell volume, 720 cc. The total white blood cell count was 10,000 with 31 per cent lymphocytes, 4 per cent stab neutrophils, 60 per cent segmented neutrophils, 2 per cent eosinophils, and 3 per cent monocytes. A stool guaiac test was positive for occult blood. Urinalysis was negative except for 3 to 10 leukocytes and 0 to 5 erythrocytes per high power field. Prothrombin time was 81 per cent.

Radiographic studies. On May 28, a barium enema was negative. On May 31, an upper gastrointestinal x-ray examination was interpreted as follows: The esophagus was negative. The stomach was normal in size and position. There was a persistent defect in the middle third of the stomach which was most pronounced on the greater curvature and in which there was a crater. The duodenum was normal in contour. Some barium was visible in the colon which was retained from the previous examination. A series of films was made for study of the small intestine. The small intestine appeared normal. There was normal motility through the stomach and small intestine and no evidence of obstruction. In the two-hour film the stomach was completely emptied and the head of the meal was in the ascending colon. Impression: probable malignant ulcer or neoplasm in the middle third of the stomach.

FIG. 1. Roentgenogram showing the persistent defect in the middle and distal portion of the stomach. Note the crater filled with barium on the proximal aspect of the filling defect.

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Hospital course. The patient was prepared for operation by being given whole blood transfusions, packed erythrocytes, and other supportive measures. On June 3, exploratory laparotomy and gastrotomy were performed. On the distal third of the greater curvature of the stomach just proximal to the pyloric antrum, there was a 2-cm. diameter, 3-cm.-high submucosal lipoma. At the apex of the lipoma there was an ulcer about 1.2 by 0.8 cm. with an 0.8-cm. depth. A partial gastric resection with removal of the lipoma and with a border of several centimeters of normal stomach was performed. The patient's postoperative course was excellent without complications. She was discharged on June 13.

Pathologic report. Gross: The specimen consisted of a previously sectioned polypoid mass removed from the stomach wall and covered by mucosa. The mass projected approximately 3 cm. above the surrounding mucosa and was 2 cm. in diameter. On the mucosal surface there was a 1.2 by 0.8 cm. ulceration; otherwise, the mucosa was not unusual. On section, the mass itself was found to be composed of a well circumscribed fatty tumor having the gross appearance of a lipoma.

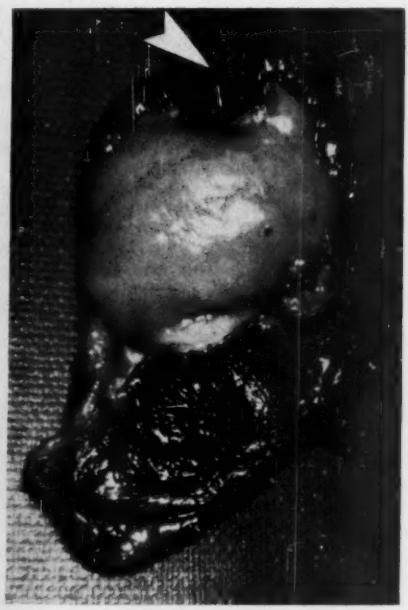

FIG. 2. Sectioned tumor which is in the *upper half* of the photograph and adjacent stomach which is in the *lower half*. The tumor mass projected 3 cm. above the adjacent stomach wall, and was 2 cm. in diameter. On the mucosal surface, the *arrow* indicates a 1.2 cm. by 0.8 cm. ulcer with a depth of 0.8 cm.

FIG. 3. Top view of the gastric lipoma before sectioning. In the center of the specimen the large ulcer is visible.

Microscopic: Section of the gastric lesion revealed a mass of fatty tumor made up of rather large, fat distended cells and scattered among these were groups of immature fat cells and proliferating fibroblasts. The mucosal surface showed a large area of ulceration which was covered by fibrin and purulent exudate. The mucosa at the edges of the ulcer showed some mild polypoid hyperplasia. There was no evidence of malignancy.

Diagnosis. Submucosal lipoma of stomach.

Final diagnosis. Submucosal lipoma of the stomach with ulceration and severe hemorrhage.

Operation. Partial gastric resection with removal of submucosal lipoma.

DISCUSSION

Gastric lipomas occur in patients with an average age of 54 years, approximately the same as for other benign and malignant gastric tumors.[8] Gastric lipomas occur with equal frequency in both sexes.[3, 9]

In a survey of 61 cases in the literature, less than half of these reported cases were incidental

findings at autopsy or in a stomach surgically removed for another lesion.[5]

In those patients presenting themselves with gastric complaints, the symptoms may be caused by mechanical interference of the lipoma with gastric emptying, by ulceration of the mucosa over the tumor, by peritoneal irritation, or by hemorrhage. Bleeding occurs rather frequently, may exhibit itself as chronic slow blood loss, or may be severe and exsanguinating.[3] In a series of 103 cases collected from the literature by Palmer,[6] 47 per cent of the patients had manifestations of hemorrhage, 27 per cent had abdominal pain, and 20 per cent had obstructive symptoms.

The roentgenologist may sometimes be able to differentiate a lipoma from a malignant tumor of the stomach since the usual lipoma is smooth in outline and may be freely movable if in the submucosa. The radiolucent shadow given by a fatty tumor is sometimes sufficient to permit the preoperative presumptive diagnosis of lipoma.[3]

Lipomas may form polypoid projections into the gastric lumen simulating leiomyomas; or, if they develop outside the muscle coat, the fatty mass can extend outward from the stomach.[7] As occurred in our cases, the lipoma has been present in the submucosa in 94 per cent of reported cases and in the antrum in 69 per cent.[9]

The decision as to what type of surgical procedure to do at laparotomy may be a difficult one. If the diagnosis of benign gastric lipoma can be made with absolute certainty, local excision is adequate for removal.[7] If there is a question of malignancy, then diagnostic help from the hospital pathologist may be necessary.

SUMMARY

A case of gastric submucosal lipoma with a clinical picture of upper gastrointestinal hemorrhage is reported and discussed. Radiographically, this lesion appeared possibly to be a carcinoma.

The tumor was removed by partial gastrectomy without difficulty, and the patient had an uncomplicated postoperative course.

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NONPENETRATING ABDOMINAL TRAUMA WITH INJURY TO BLOOD VESSELS

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During a review of the cases of nonpenetrating trauma to the abdomen at the Colorado General Hospital, we were impressed by some of the vascular phenomena that had occurred. An investigation of the literature was then made for other cases of vascular injury due to nonpenetrating trauma. This paper briefly mentions the reported cases, and adds our experience to that list.

There are but few documented cases of blunt injury to intra-abdominal vessels. Kidd[1] collected 10 cases of vena cava injury and added a case of his own, but only two were due to nonpenetrating abdominal trauma. Both of these cases recovered. These are the only two cases we can find reporting of veins of the abdomen damaged by blunt injury. One case was reported in 1954 by Ulvestad,[2] of injury to the superior mesenteric artery. The vessel was sutured and the patient recovered. No other cases of nonpenetrating vascular injury were found.

Six cases of blunt abdominal trauma with vascular injury were found at the Colorado General Hospital. A short summary of each case will be presented here, and a brief note will follow in an effort to elucidate the points which may be gained from our experience (table 1).

CASE REPORTS

Case 1. S. A. J. (CGH-100627). This 2-year-old white child was run over by a car. He vomited blood, became semicomatose, and was sent to Colorado General Hospital about one hour after injury. He was pale, lethargic, had a blood pressure of 90, pulse of 150, and labored respirations 30 times per minute. The abdomen was tender on the right side and distended. An area of ecchymosis was present in the right lower quadrant. Paracentesis yielded gross blood. At operation, the dome of the liver was avulsed from the diaphragm. A large rent in the right hepatic lobe was sutured; however, bleeding persisted massively from the right hepatic vein and vena cava from which it was avulsed. Exposure was difficult and hemorrhage continued. Cardiac arrest ensued and all measures for resuscitation failed. The child died.

Case 2. E. K. H. (CGH-55599). This 21-year-old sailor was thrown from his convertible. He was alert and had no complaints upon arrival in the emergency room over an hour later. During observation, he became acutely ill with abdominal pain, and went into shock. His abdomen was diffusely tender and became rigid and board-like within 2 hours. Vasopressors and 2000 ml. of whole blood did not reverse his shock. At laparotomy, massive bleeding was noted from a severely lacerated spleen and liver. A third of the right lobe of the liver was free in the abdominal cavity, and another third was almost hanging free. Other lacerations necessitated the attempt to a right hepatectomy. The spleen was removed. Hemorrhage was also coming from the vena cava at the site off which the right suprarenal vein had been avulsed. During the attempt at hepatectomy, the patient went into cardiac arrest. He died despite cardiac massage and blood replacement.

Case 3. R. K. (CGH-107686). This 17-year-old man was admitted soon after being pinned under the cab of a truck. He complained of chest and abdominal pain. Examination revealed pallor, hypotension, and a rapid thready pulse. The chest was clear but his abdomen was diffusely tender and distended. The diagnosis of abdominal bleeding was made. Shock continued despite cut down. He was taken immediately to the operating room where laparotomy revealed massive hemorrhage from the liver. The avascular left lobe was excised and packing controlled the lacerations. However, bleeding under the liver continued from avulsion of the hepatic veins off the vena cava. Cardiac arrest interrupted suturing of the rents in the vena cava. Cardiac massage failed and the patient died.

These 3 cases illustrate severe associated injuries to the liver and all the patients were in shock at operation. The authors are aware of no cases of successful repair of the intrahepatic vena cava or avulsed hepatic veins. In case 2, the hemorrhage from the suprarenal vein and avulsion site from the vena cava was just one

TABLE 1

Documented cases of nonpenetrating abdominal trauma

Author and Case No.	Date	Age/Sex	Mechanism of Injury	Preoperative Diagnosis	Time Before Operation	Vascular Problem	Other Injuries	Operative Treatment	Result	Comment
Kidd 2	1922	Young/M	Fall—no wound	Not given	Not given	Rent in left side of vena cava	None	Suture of rent	Recovery	
Kidd 5	1926	41/F	External violence	Not given	10 days	Rent just above beginning of cava	Ruptured into an ovarian cyst	Ligation of vena cava	Recovery	
Ulvestad 1	1954	Young/M	Pinned under tailgate of truck	Not given	Not given	Laceration of superior mesenteric artery	Laceration of bowel. Blue bowel 18 in. from ligament of Treitz to middle of transverse colon	Repair lacerations of artery and bowel by suture. Tied off superior mesenteric vein	Recovery	Occasional diarrhea in postoperative course
Shuck & Trump 1	1957	2/M	Abdomen run over by auto wheel	Laceration of liver	2½ hr.	Right hepatic vein avulsed from vena cava	6 in. laceration of liver. Liver avulsed from dome of diaphragm	Suture attempted but unable to control caval bleeding. Cardiac massage	Death	Severe liver and inadequate exposure of hepatic vein and vena caval bleeding
Shuck & Trump 2	1953	21/M	Thrown out of convertible when it rolled over	Ruptured spleen, liver and possible left kidney	7 hr.	Avulsed right suprarenal vein from vena cava	Multiple severe liver lacerations. Multiple spleen lacerations. Fracture right clavicle	Splenectomy. Clamped hepatic artery and portal vein. Right hepatectomy tried. Cardiac massage	Death	Liver unsalvageable from severe injury. Bleeding cava and suprarenal vein were only contributory
Shuck & Trump 3	1958	17/M	Crushed under 3-ton truck	Abdominal bleeding	1½ hr.	Avulsed hepatic veins from vena cava	Liver lacerations. Avascular left lobe of liver	Excision left lobe of liver. Packed lacerations clamped above and below caval rents. Cardiac massage	Death	Because of exposure probems and massive bleeding, unable to control caval bleeding

Shuck & Trump 4	1959	47/M	Thrown from car	Ruptured abdominal viscus. Probably bowel	38 hr.	Avulsion and thrombosis of left colic vessels	Avulsion of descending colon with necrosis. Fracture of right humerus. Rib fractures	Hemicolectomy. Double colostomies. Tracheostomy	Recovery	Bowel reapproximated and continuity established at second operation 3 months later. Normal movements now
Shuck & Trump 5	1958	54/M	Chest hit by wheel	Ruptured spleen	1½ hr.	Avulsion of 2 branches off superior mesenteric vein—2 hole	Fractured 4th left rib	Suture of rents in superior mesenteric vein—clamped and ligated the two branches	Recovery	Developed delirium tremens on 3rd postoperative day. Otherwise normal course
Shuck & Trump 6	1950	43/M	Thrown from car when hit by train	Multiple fractures	51 hr.	Laceration of splenic vein at hilus	Lacerated spleen. Fracture of pelvis. Compound fractured femur; fractured fibula; dislocation of tibia	Died on way to operating room	Death	Abdominal trauma not suspected until few hours before death; died on cart to operating room

other severe intra-abdominal injury. These 3 cases were very complex, and there appears to be no simple answer to the problem.

Case 4. A. V. (CGH-122841). This 47-year-old man was seen 24 to 30 hours after being thrown from his car. His local physician kept him in a small hospital overnight. Examination revealed an obvious fracture of the right humerus and he complained of diffuse abdominal pain and chest pain. The blood pressure was 92/70, pulse 120, and respirations were 44. He was pale, acutely ill, dehydrated and lethargic. Chest and abdomen were tender diffusely. X-rays showed many rib fractures, humerus fracture, but no free abdominal air. At laparotomy, a putrid odor came from the abdomen and the brown fluid present was cultured and found to be sterile. The descending colon was black and detached from the posterior abdominal wall. No perforation was found, but the left colic vessels were avulsed from the mesentery and thrombosed. A large retroperitoneal hematoma was not disturbed. The necrotic bowel was resected, and the splenic flexure and sigmoid were brought out into the abdominal wall where colostomies were secured. The postoperative course was prolonged by an ileus, but recovery was complete. Four months later the bowel was anastomosed, re-establishing its continuity. The patient is now having normal bowel movements.

This was a case of avulsion of the descending colon off the posterior abdominal wall with severence of the blood supply without bleeding. Thrombosis was probably immediate. The lack of bowel rupture was also a striking finding in the face of the severe injury. No similar cases were found in our literature review.

Case 5. J. H. (CGH-107554). This 54-year-old man was admitted immediately after being hit in the chest by his steering wheel during an auto accident. He was rational, anxious, pale and complaining of abdominal pain. Blood pressure was 60/0, and pulse was 120. The abdomen was flat, diffusely tender and no bowel sounds were heard. Right lower quadrant paracentesis yielded gross blood. Diagnosis of a ruptured spleen was made. A Levin tube was inserted, and blood was given through cutdowns. Blood pressure remained low and the patient was taken to the operating room. Laparotomy revealed intact liver, spleen and bowel. Bleeding was arising from 2 rents in the superior mesenteric vein off wh[illegible]a 2 tributaries were torn. The holes were sutured, and the free ends of the veins were found and ligated. His postoperative course was complicated by full blown delerium tremens on the third day. He responded to treatment and was discharged on the tenth day in good condition. Follow-up 2 months later revealed no complications.

This is an example of a ruptured vein with no other injury within the abdomen. He responded well to blood replacement and early surgical intervention. Such a diagnosis preoperatively is practically impossible, but if intraperitoneal hemorrhage is suspected, and the surgeon is skilled in vascular techniques, the exact site of bleeding becomes less important.

Case 6. H. R. (CGH-27958). This 43-year-old farmer was thrown from a car that was hit by a train. On arrival in the emergency room he complained of pain in his legs. Blood pressure was 64/50, pulse 120 and thready and the patient was alert. Multiple, bilateral, lower extremity compound fractures were obvious. There was a hematoma of the right lower quadrant of the abdomen and of the genitalia. Hypotension persisted despite blood plasma and fluid administration. The fractures were debrided, irrigated and dressed. Some time later, shock recurred and urinary output ceased, although a total of 5000 ml. of whole blood had been given. His abdomen became tense and tender and his condition deteriorated. Surgical consultation was obtained and the patient was taken to the operating room after the diagnosis of internal injury was made. The patient died on the way to surgery, 61 hours after his injury. Autopsy revealed a ruptured spleen and 400 ml. of intraperitoneal blood. However, the splenic vein was torn near the hilus and massive retroperitoneal hemorrhage was disclosed.

This case points out how serious skeletal injuries and their urgent treatment may delay the diagnosis of an acute abdomen and its correction. Whether an immediate exploration would have effected a good result will remain an item for conjecture; however, prolonged hypotension contributes so greatly to renal shutdown that exploration is probably indicated if blood replacement does not correct the hypotension in the face of possible abdominal trauma.

SUMMARY

The literature was reviewed for cases of vascular injury due to blunt abdominal trauma, and

only 3 such cases were found. All these patients survived. Perhaps similar wounds have been sustained, but not reported because of death without a diagnosis. Also, other surgeons may not have reported their experience with such accidents. Thus it is difficult to appreciate the incidence of this type of injury.

Six cases of blunt abdominal trauma to blood vessels have been collected at the Colorado General Hospital within the past 10 years. Case reports have been presented and the salient features of each case have been discussed. It is felt that such vascular injuries may be more frequent than hitherto suspected.

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CARCINOMA OF THE THYROID: SURGICAL ASPECTS

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Most of the malignant tumors of the thyroid are slow growing. Some of them seem to lie dormant for many years, then begin to grow more rapidly and metastasize widely. These tumors are not frequently seen by any one practitioner, whether he be an internist, general practitioner or a general surgeon. The rarity and slow growth of these tumors account for a rather indifferent attitude of many practitioners toward the surgical treatment of nodular goiter and carcinoma of the thyroid; however, it is a well documented fact that patients do die from these tumors, more frequently from local extension or mediastinal metastases, but also occasionally from distal metastases to the spine or brain. The low incidence and long follow-up periods required make it extremely difficult to obtain mortality rates and also to evaluate therapy. It is evident that a 5-year follow-up is inadequate in reporting both results of treatment and mortality in this disease. It seems obvious that the more effective management of carcinoma of the thyroid depends upon a more aggressive attitude of the family physician, internist and surgeon toward the diagnosis and surgical removal of nodular goiter, particularly the solitary nodule.

The following discussion results from a study of 19 private cases operated upon by Dr. John H. Wootters of Houston and me. These were encountered in a series of 309 thyroidectomies.

PATHOLOGY

The over-all incidence of carcinoma of the thyroid in this series is 6.1 per cent. It is 4.2 per cent in regard to multinodular goiter, 12.5 per cent in the solitary nodule (preoperative) and 25 per cent of the true neoplasm.

The etiology of the thyroid cancer is not known. It now seems more likely that this disease in many cases begins as a cancer rather than arising in adenoma or in any type of existing goiter. A number of cases have been reported in younger patients who have had external irradiation to the neck in childhood, often for an enlarged thymus, but also for other conditions. A relationship is strongly suggestive; however, a further observation is necessary to prove this. Hormone relationships have also received considerable attention recently. There is evidence that the thyroid-stimulating hormone of the pituitary will stimulate normal thyroid and also functioning metastatic tissue, which, in turn, means that hypothyroidism can possibly stimulate metastatic tissue to grow.

We have taken the following classification, which is a slight modification of that used by Warren and Meissner, as the basis for our discussion:

I. Differentiated carcinoma
  A. Follicular carcinoma
    1. Low grade localized carcinoma and follicular adenoma
    2. Follicular adenocarcinoma
  B. Papillary carcinoma
    1. Low grade localized carcinoma and papillary adenoma
    2. Papillary adenocarcinoma
  C. Hürthle cell carcinoma
II. Undifferentiated carcinoma
  A. Small cell carcinoma
  B. Giant cell carcinoma
III. Miscellaneous
  A. Fibrosarcoma
  B. Epidermoid carcinoma
  C. Lymphoma
  D. Metastatic carcinoma

Papillary tumors make up over 50 per cent of the thyroid carcinomas encountered clinically. These tumors often manifest contralateral foci which represent either intragland metastases or multiple foci of origin. They are also often associated with contralateral involvement of the cervical nodes. They usually spread only by the lymphatics, but may occasionally metastasize by blood.

The follicular tumors may also occur as localized carcinoma in an adenoma or as follicular adenocarcinoma. These tumors are more apt to metastasize by the blood stream, particularly to the bones and to the lungs; however, they also metastasize by the lymphatics. The metastases tend to be present in the regional lymph

nodes located above and below the isthmus of the thyroid to the nodes in the tracheoesophageal groove, to the jugular nodes, to the posterior triangle, and occasionally to the submaxillary triangle. The studies by Rouvière[33] pertaining to lymphatic drainage of the thyroid and also the work of McClintock and his associates[23] indicate that the superior mediastinal nodes are involved in a significant number of cases. The undifferentiated and miscellaneous tumors are fortunately rare.

CONTROVERSIAL VIEWPOINTS

Crile[7-9] takes a more conservative viewpoint. He advocates an adequate excision of the primary tumor along with careful dissection of the centrally located nodes of the superior mediastinum and paratracheal nodes behind the thyroid and along the trachea. He states that patients with papillary carcinoma die from invasion of the trachea, esophagus and mediastinum by the primary tumor and its centrally located nodes, and not from uncontrolled lateral cervical metastases. He feels that papillary tumors are amenable to hormone control. His arguments and statistics to support his viewpoint are very convincing. He has demonstrated that primary operations which cut across tumor tissue have increased by tenfold the possibility that the patient will have recurrent local or metastatic disease.

Beahrs and Woolner[2] reported in the January 1959, issue of *Surgery, Gynecology and Obstetrics* an experience of 136 cases of papillary carcinoma managed by the more conservative approach, with only five radical neck dissections done. Their results are summarized in table 1. This study emphasizes two important aspects of this problem: (1) Properly planned conservative operations give good results. (2) Long time follow-ups are difficult to obtain.

Frazell and Foote[14-16] of Memorial Hospital are among the group of surgeons who advocate a more radical approach to this problem, including radical neck dissection prophylactically. The thinking of these men is probably greatly influenced by the type of material which they have had for study. Many of their patients represented treatment failures. Many have had inadequate primary treatment; therefore, it would seem logical that their thinking would tend toward a more radical early treatment of the disease. This viewpoint would be supported by the pathologic findings of 61.2 per cent node involvement in clinically negative patients. Studies by other careful workers[5, 6] supported these findings; however, it is interesting to note that Frazell and Foote,[15] in a recent 25-year survey published in *Cancer*, 1958, make the following statement: "It is evident that there is no statistical proof of the superiority of the radical surgical treatment of papillary carcinoma over the conservative approach." They also further state: "This experience fails to settle the issue of whether or not routine radical neck dissection for metastatic papillary carcinoma will be a deciding factor in the ultimate survival of the patient."

TABLE 1

Papillary carcinoma: 136 patients total[2]

Survival Period	Traced	Living	Per Cent	Undetermined
5-year	133	129	97	3
10-year	124	109	87.9	12
15-year	49	37	75.5	8

In cases where there is extensive metastatic node involvement, McClintock and his coworkers[23] do a total thyroidectomy plus a superior mediastinal dissection, using a sternal splitting incision. The Lahey group[6] do prophylactic radical neck dissections and, if positive nodes are present, give super voltage x-ray therapy.

The preponderance of the evidence at this time seems to indicate that the adequate primary operations, that is either total lobectomy or total thyroidectomy along with limited neck dissection, give better results than the more radical operations and that prophylactic radical neck dissections are not necessary.

TREATMENT

Surgery. When we operate upon a patient with a multinodular goiter, we carefully examine both lobes and the isthmus and also patiently search for involved lymph nodes above and below the isthmus, above the sternal notch and in the tracheoesophageal grooves. A subtotal resection is done and the specimen given to the pathologist for examination, including frozen section if indicated.

If unilateral gross pathology is present, the contralateral lobe is exposed by anterior and lateral dissection; it is then carefully examined and searched for nodes. If the contralateral lobe

is grossly normal and no nodes are found, the involved lobe, along with the isthmus, is removed and submitted to the pathologist for frozen section. If a frozen section diagnosis of carcinoma is made, a total thyroidectomy is done. A total thyroidectomy is done in the papillary group because foci of carcinoma are often present in the opposite lobe. It is done in the follicular carcinomas because we desire to do tracer studies on these patients and ablate any normal and abnormal thyroid functioning tissue with radioactive iodine.

If a diagnosis cannot be made at frozen section and the permanent sections are reported as follicular or papillary carcinoma, we do not reoperate upon these patients, but keep them under careful observation. If involved nodes are seen, we advise dissection of the area between the jugular veins and between the notch in the thyroid cartilage above and the sternal notch below. Radical neck dissections are done only when there is extensive gross involvement of the cervical nodes and when nodes become manifest after total thyroidectomy and ablation with I^{131}.

Radioactive iodine. We have had the good fortune to be near an excellent radioisotope department. Dr. Herbert Allen has been extremely interested in this disease with its controversial therapeutic aspects, and also Dr. Carleton M. Neil, both of Houston. We have carried out tracer studies on our patients four weeks after surgery. We have found that the most carefully performed total thyroidectomies are seldom anatomically complete and thyroid functioning tissue is found near the operative area. This has been ablated with radioactive iodine. If metastases are demonstrated, these are surgically removed or treated with I^{131}, depending upon their location and affinity for I^{131}. These patients are then placed on adequate doses of exogenous thyroid extract to maintain them in a euthyroid state. Our follow-up studies include uptake and gammagrams at varying intervals, particularly in those patients who have had total thyroidectomies. The preoperative demonstration of a hypofunctioning nodule is an indication for

TABLE 2

Review of private cases

Patient	Sex	Age	Year	Type	Treatment
L. W.	F	36	1948	Papillary	Total thyroidectomy
L. C.	F	33	1949	Papillary	Total lobectomy
V. J.	F	35	1951	Alveolar	Total lobectomy, x-ray therapy
M. R. W.	F	41	1953	Undifferentiated	Total thyroidectomy, R.I.* therapy, thyroid
L. M.	F	11	1953	Papillary	Total lobectomy, x-ray therapy
G. C. S.	M	38	1953	Papillary	Total thyroidectomy, neck dissection, R.I.* therapy (100 mc.), thyroid
J. H.	F	16	1953	Papillary	Total lobectomy
M. W.	F	38	1956	Mixed, papillary and follicular	Total thyroidectomy, neck dissection, R.I.* therapy, thyroid
F. N.	F	30	1957	Mixed, solid and follicular	Total thyroidectomy, R.I.* therapy (100 mc.)
F. W.	F	40	1958	Follicular	Total thyroidectomy, x-ray therapy (6500 r), thyroid
E. K.	F	48	1958	Mixed, papillary and follicular	Total thyroidectomy, R.I.* therapy (92 mc.), thyroid
Z. B.	F	27	1958	Follicular	Total thyroidectomy, R.I.* therapy (169 mc.), thyroid
L. McD.	F	48	1959	Mixed, papillary and follicular	Total thyroidectomy, thyroid
M. W.	F	38	1959	Follicular	Total lobectomy, right subtotal, left, R.I.* therapy (25 mc.), thyroid

* Radioactive iodine.

surgery, since this lesion is more likely to be malignant.

External irradiation. We have used external irradiation in the tumors of low grade malignancy only when there was evidence at the time of surgery that the tumor had extended beyond the thyroid; that is, adherence of the ribbon muscles or involvement of the trachea. It is also indicated when the primary lesion is not resectable and as a palliative measure in the undifferentiated and miscellaneous groups. It is probably most effective in the lymphomas. It is difficult to find objective evidence regarding the effect of adequate external irradiation on the metastatic nodes of the papillary and follicular tumors. More information of this type is necessary before the true status of this method in the treatment of carcinoma of the thyroid can be determined.

Hormone considerations. It has already been indicated in this discussion that evidence has been presented during recent years that the growth of some of these tumors is encouraged by the thyroid-stimulating hormone (TSH) and, conversely, that the growth of some of these tumors is retarded by feeding exogenous thyroid extract to suppress the formation of TSH. This is an interesting concept and may prove to be extremely useful; however, further time is necessary for its evaluation. We give 3 grains of thyroid extract a day. We have found only one patient who will not tolerate this amount (table 2).

SUMMARY

1. The nodular goiter should be removed unless risk is prohibitive.

2. The discrete nodule should be removed with the involved lobe and isthmus for tissue examination.

3. The contralateral lobe should be carefully exposed and examined at the time of surgery.

4. Involved nodes should be searched for carefully.

5. Frozen section diagnosis is very important. It should always be attempted.

6. If frozen section is positive, total thyroidectomy is done with limited neck dissection if indicated.

7. If gross extensive lymph node involvement is present, radical neck dissection is done, preferably at the first operation.

8. If nodes develop after total thyroidectomy, a radical neck dissection is done.

9. Prophylactic radical neck dissection is not advised.

10. Residual thyroid tissue or metastatic functioning tissue is ablated with I^{131}.

11. If there is evidence of local extension of the primary neoplasm, external irradiation is also given.

12. Thyroid extract (3 grains) is given after surgical excision for carcinoma to suppress thyroid-stimulating hormone.

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EXPERIMENTAL TRIPHASIC HEPATOGRAPHY: AN EXPERIMENTAL STUDY[a]

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Splenoportography with intrasplenic pressures has now become solidly entrenched as a diagnostic aid for hepatic and extrahepatic disease processes.[2, 4, 10, 13] Since its introduction to medicine,[1, 3, 5, 8, 9] there have been only a few changes in techniques and dyes. This report concerns itself with an attempt to demonstrate triphasic x-ray visualization of the liver with splenic injection of 52 per cent Cholografin methylglucamine.[b] By selecting this material and utilizing serial x-rays, we tried to demonstrate a portal venogram, hepatogram, and cholangiogram with a single injection. An extensive review of the past five years of English and foreign medical literature and correspondence with the distributing company[7] seems to indicate a need for such a study.

METHOD

Large mongrel dogs were anesthetized with pentobarbital sodium.[c] There was no selection as to sex or age of the animals. No premedication was used. A cuffed endotracheal tube was inserted and intermittent positive oxygen pressure was administered at the rate of 20 to 30 per minute. A few of the animals were done under moderate (30°C.) hypothermia. Preliminary studies of the biliary tree anatomy in the dog were done by dissection and direct injection with dye (fig. 1). In the early part of the study, the spleen was delivered through a midline upper abdominal incision. Direct injections were made into the pulp at the hilum with 10 to 20 cc. of dye. Later, this was done by percutaneous puncture. Various dyes and mixtures of dyes were used including Cardiografin 85 per cent,[d] Cholografin methylglu amine 52 per cent and Renografin 76 per cent.[d] X-ray exposures were taken immediately at the inclusion of injection and for various periods up to two hours. A few animals were also studied by intravenous injections of Cholografin and direct injections into the biliary system.

FINDINGS

Of 28 dogs, we performed satisfactory hepatograms on 6 and cholecystogram and/or choledochogram on 3. With close attention to technique, portal venograms were regularly obtained. Figure 2 is a typical portal venogram with intrasplenic injection of Cholografin in a dog. We were disappointed and perplexed by our subsequent inability to demonstrate well either the "hepatic" phase or the "cholangiographic" phase of liver visualization.[12] The hepatic phase when present was represented by transient liver staining and usually seen in the "immediate" x-ray exposures. Only on a few occasions was it felt to be of sufficient contrast to be of diagnostic importance (fig. 3). Biliary tree visualization was faint in the few instances where it was thought to be present (fig. 4). Subsequently, several spleens were sectioned under the microscope. There was no apparent change in these organs after injection of the dye. Intravenous Cholografin was no more rewarding than intrasplenic Cholografin in terms of visualization of the biliary tree.

DISCUSSION

Cholografin would seem to represent an ideal agent for splenoportography. With this dye one could expect to visualize not only the portal venous system, but also the hepatic parenchyma and the biliary drainage system. Studies with I^{131}-tagged Cholografin in dogs and humans seem to indicate a lessened affinity in dogs for this dye.[11] It may be that the dog and human differ considerably in this regard. We have encountered

[a] From the Surgical Research Laboratory of the Creighton Memorial St. Joseph's Hospital, Omaha, Nebraska. Supported by a grant from the Nebraska Heart Association.

[b] Iodipamide methylglucamine, 52 per cent, supplied by the Squibb Institute for Medical Research (52 per cent solution of the methylglucamine salt of N,N′-adipylbis-[3-amino-2,4,6-triiodo] benzoic acid).

[c] Veterinary Nembutal sodium supplied by Abbott Laboratories.

[d] Supplied by the Squibb Institute of Medical Research.

FIG. 1. This is a cholangiogram after direct injection of 40 cc. of radiopaque dye into the gall bladder. Note the midline position of the gall bladder and the slanting course of the common duct to the duodenum. There is slight soiling of the peritoneal cavity with dye.

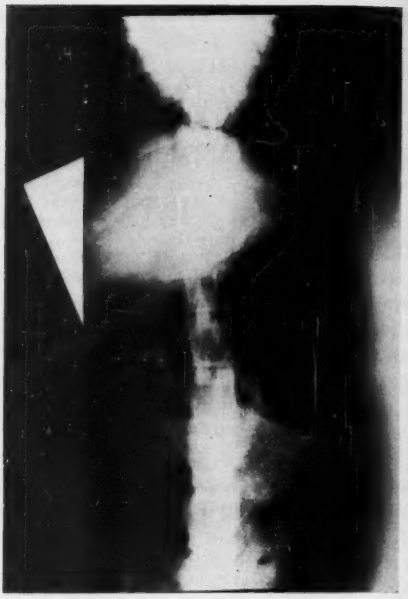

FIG. 3. Hepatogram 30 sec. after intrasplenic injection of 10 cc. of Cholografin. There is faint visualization of the common duct (indicated by *arrow*).

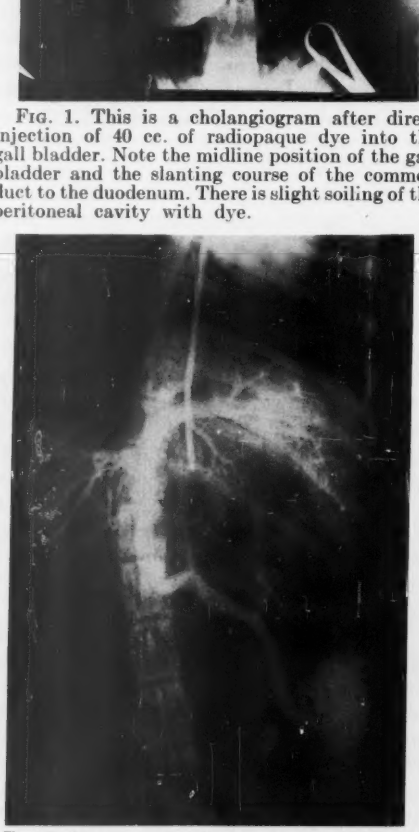

FIG. 2. An x-ray reproduction of a typical portal venogram. It is noteworthy that the portal vein follows a course similar to that seen in humans. Telethermometer is seen in the esophagus or stomach. This is a delayed exposure taken 1 min. 15 sec. after the injection of 20 cc. of Cholografin under hypothermia.

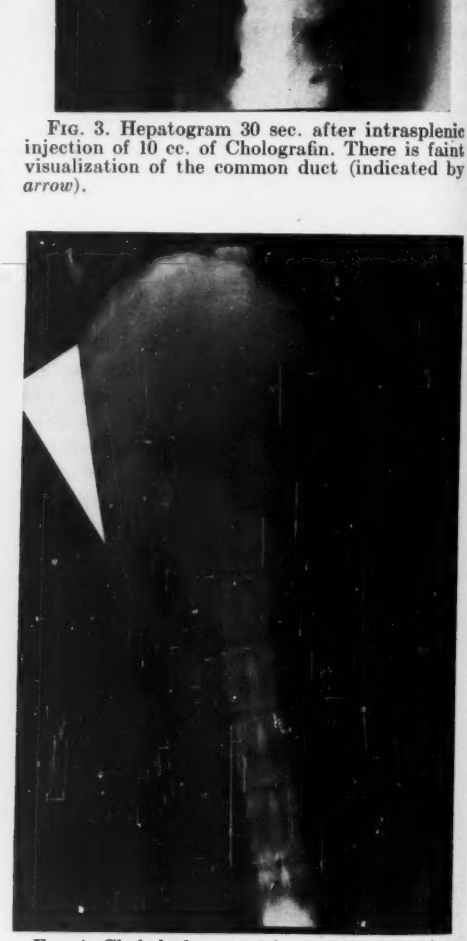

FIG. 4. Choledochogram 2 hours after injection of Cholografin (indicated by *arrow*)

no experimental study of attempts to visualize with x-ray the biliary system in dogs by this method. Despite these results, we feel that a limited trial of this method in humans is indicated and we are implementing this at the present time.

The use of Cardiografin and Renografin for splenoportography proved satisfactory. Pyelograms were obtained in the dogs in which Renografin was used. In many of the portograms there was evidence of "streamlining"[6] in the dog's portal vein, particularly where the superior mesenteric vein enters. We demonstrated no late phases in these animals.

We are continuing to investigate iodinated materials as used in splenoportography. Certainly iodine containing emulsified fat injected into the spleen should give a vivid hepatogram. We are also considering a study of the secretion of Cholografin into the biliary tree of dogs.

SUMMARY

A preliminary report of attempts to perform splenoportocholodochography in dogs utilizing Cholografin methylglucamine, 52 per cent, is reported. In only 3 of 28 animals were we able to demonstrate a cholangiographic or choledochal phase of secretion. A trial in humans is under investigation.

Acknowledgment: We are indebted to Dr. D. Arnold Dowell and his staff in the Department of Radiology at St. Joseph's Hospital for their aid in the performance of this experimental study.

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INTRA-ABDOMINAL LYMPHOID HYPERPLASIA AND APPENDICEAL DISEASE

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A survey of 100 consecutive appendectomies during the years 1952 to 1957 validates the changing nature of appendiceal disease, with evidence of the increasing importance of a syndrome created by intra-abdominal lymphoid hyperplasia primarily appendiceal. Within recent years, a disease, formerly recognized as mesenteric adenitis, has come to be closely linked with appendiceal disease. For the surgeons practicing during the early part of the century, before the widespread use of antibiotics, mesenteric adenitis, if ever clinically diagnosed, was considered a medical disease. It was not to be treated surgically or if an appendix were inadvertently removed with lymphoid hyperplasia, the surgeon or pathologist would be inclined to place this case in the chronic category.

Other problems related to appendiceal disease occur when a clinical diagnosis of acute disease results not in appendectomy but in resection of involved tubo-ovarian tissues, closure of perforated ulcer, resection of infarcted appendices epiploicae, or even cholecystectomy.[5] At other times, a carcinoid[6] of the appendix might be removed in the course of an incidental appendectomy unrelated to a preoperative diagnosis of appendiceal disease. By reviewing 100 consecutive appendectomies by a single surgeon, a more accurate picture of appendiceal disease can be realized. The need for such a study has prompted the following analysis which might serve as a basis for tissue committees or other record review teams in hospitals throughout the country.

INCIDENCE

Figure 1 depicts an analysis of 100 appendectomies performed at St. Patrick's and Lake Charles Memorial Hospitals between 1952 and 1957. Seventy of 100 cases, confirmed by the pathologist, revealed either acute appendicitis or lymphoid hyperplasia of the appendix. Fifteen of the 100 cases were incidentally removed secondary to other surgery, and revealed no unusual disease. A remaining 15 per cent were reported by the pathologist as showing no acute inflammatory reaction or lymphoid hyperplasia and were not recorded as removed incidental to other surgery. A breakdown of these 15 cases is presented in table 1 and illustrates the preoperative diagnosis with the associated condition. Table 1 further shows that 60 per cent of the acute cases were in females with symptomatic right ovarian alterations. In an additional 5 cases, the appendix was removed with a diagnosis of chronic recurrent disease and of these cases 3 had fecoliths in the appendix and 2 presented no pathologic change.

When the nonacutely diseased and incidentally removed appendices are not considered, the changing picture is revealed as seen in figure 2. The increase in lymphoid hyperplasia of the appendix is clearly illustrated in the series of appendices removed for acute disease, with an increase from 17 per cent in 1952 to 42 per cent in 1956.

PATHOLOGY

Pathologists, surgeons and pediatricians are continually impressed by the frequency of appendectomies for suspected appendicitis during periods when there are an unusual number of cases of actual acute suppurative appendicitis. In many of the suspected cases, lymphoid hyperplasia, often with associated mesenteric adenitis, is the principal finding. In the typical case, the appendix may not be unusual externally, but on sectioning it is noted that the lumen is no longer discernible. It is imperative that comprehensive microscopic study of all appendices be performed for accurate diagnosis. Two slides of patients 25 years of age contrast well the so-called normal appendix, *above*, with one presenting appendiceal lymphoid hyperplasia, *below* (fig. 3). The lower slide shows an overabundance of lymphoid follicles[1] in the submucosa the predominence of which actually causes obliteration of the lumen in

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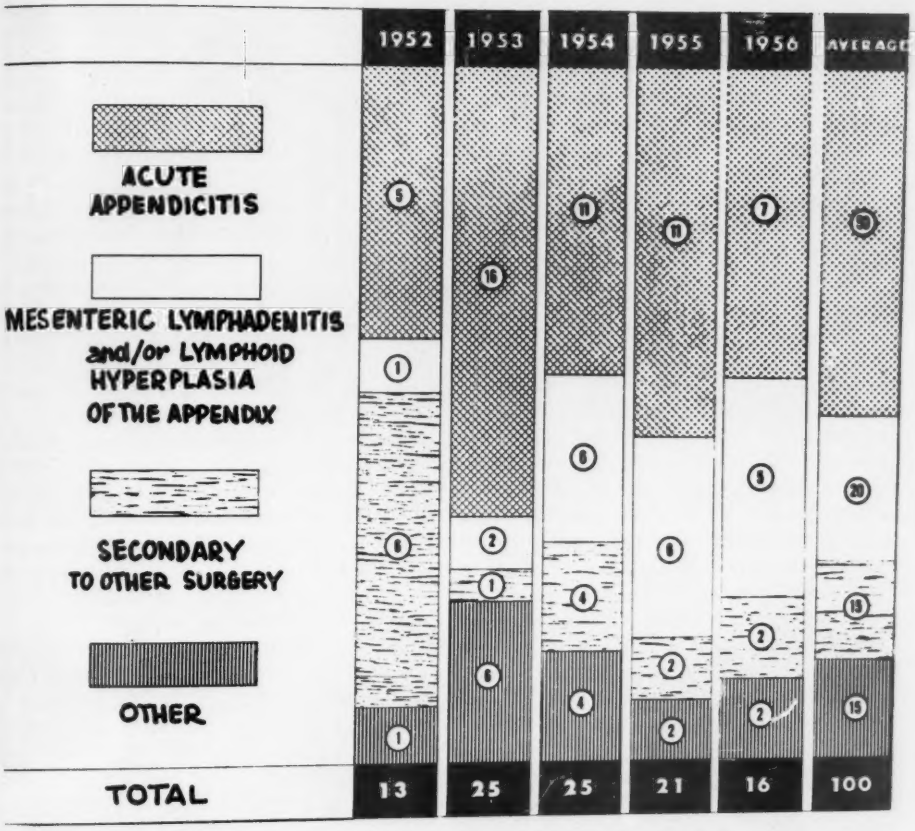

FIG. 1. One-hundred consecutive appendectomies, 1952 through 1956 (Weiss)

TABLE 1

Clinico-pathologic summary of 15 appendectomies without significant microscopic appendiceal disease: preoperative diagnosis of appendicitis

Clinico-pathologic Findings	Acute Disease	Chronic Recurrent Disease
Right tubal or ovarian findings	6	
Appendiceal oxyuriasis	1	
Mild lymphoid hyperplasia of appendix	1	
Sigmoid diverticulitis	1	
Fecal impaction of appendix	1	
Fecalith of appendix		3
No pathologic change		2

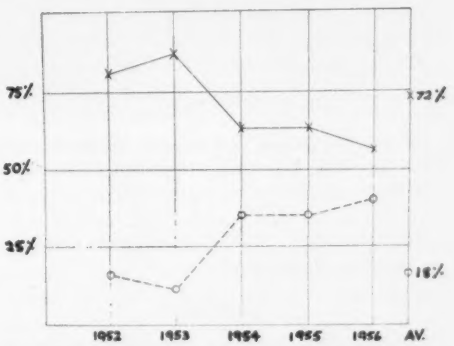

FIG. 2. Clinico-pathologic correlation of acute appendiceal disease (Weiss and Ranier). × = acute disease; ○ = mesenteric lymphadenitis and/or lymphoid hyperplasia of the appendix.

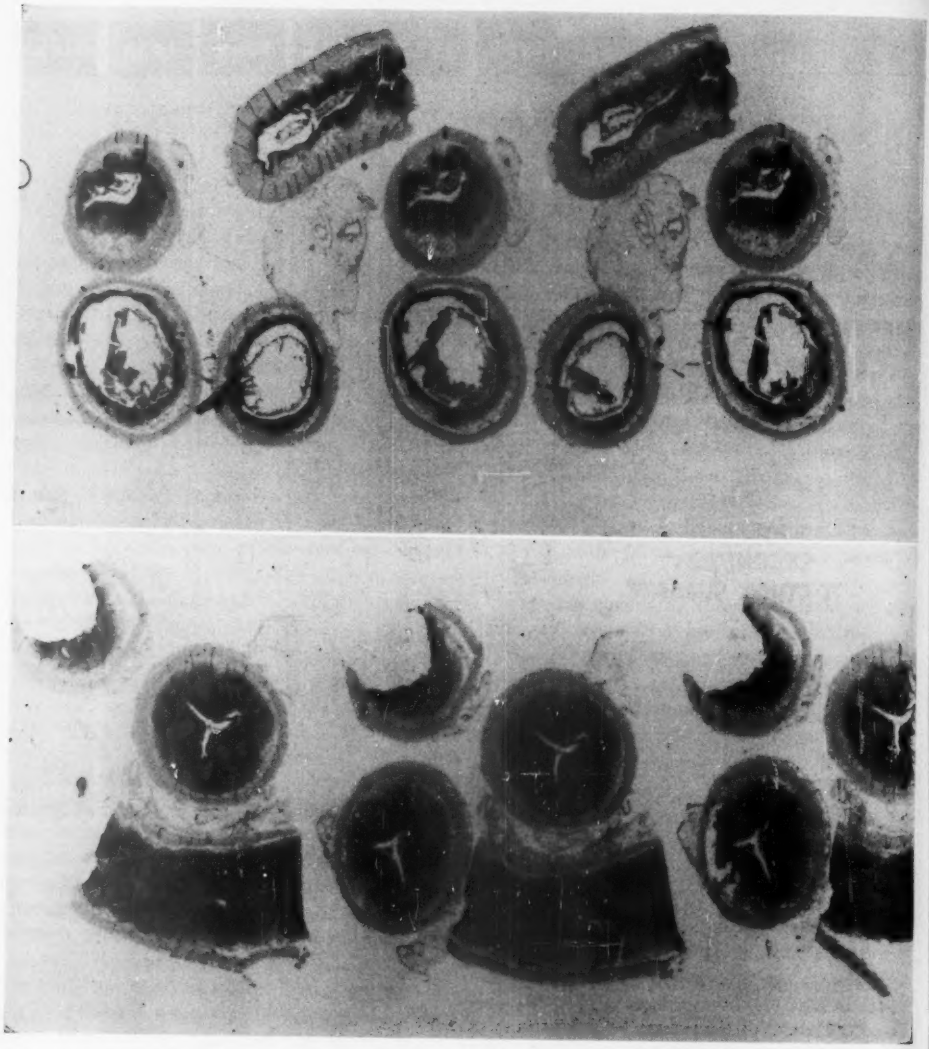

FIG. 3. Transverse and longitudinal H and E stained sections of normal vermiform appendix. *Top*, note the dark staining lymphoid tissue zone. *Bottom*, compare dark staining of follicular zone with above.

fresh tissue. The apparent small lumen is created by fixation shrinkage.

DIAGNOSIS

The clinical diagnostic criteria of acute appendicitis remain unchanged. There is no substitute for a complete history, thorough physical examination and good clinical judgement.

The classical clinical criteria for the diagnosis of obstructive appendicitis are seen less frequently.[4] Appendiceal disease due to lymphoid hyperplasia with associated lymphadenitis has been recognized more commonly in recent years, as borne out by this study. The patients with this disease often appear in the age group between 5 and 15 years with a history of recurrent abdomi-

nal symptoms. Such cases should be thoroughly studied prior to surgical intervention. If other disease is recognized, such as intestinal parasites, allergies, or dietary indiscretions, it should be treated medically.

Occasionally the pain may be so severe or excruciating as to incapacitate for a 12- or 24-hour period. Many of these patients are above average mentally and would prefer not missing school, work or play, but because of intra-abdominal symptoms must limit their activity. When seen during the acute attack, the patient is obviously in pain with tenderness across the root of the mesentery, sometimes more severe in the left upper quadrant than in the right lower quadrant. There may or may not be rebound tenderness. Seldom is rigidity present, except for splinting due to acute abdominal pain in hyper-reactive persons. Diffuse intra-abdominal effusion, however, may at times account for extensive rigidity throughout the entire abdomen simulating diffuse suppurative peritonitis. Rectal examination will aggravate the hypersensitive patient and will not usually contribute to any localized findings. When the acute attack subsides, the patient will often have residual soreness along the mesenteric root, but seldom a combination of tenderness, rebound and rigidity in the right lower quadrant. Attacks of this nature might occur frequently during a day or may occur infrequently during a year. There is no set pattern to the attacks, although they are seen more commonly during periods of upper respiratory infections. Many times this disease is confused with an emotional disorder of psychosomatic origin.

It is helpful to recall in diagnosis of lymphoid hyperplasia the text of Lewis[3] with regard to the production of pain. Lewis points out the summation effect of nerve impulses, stating "that several subthreshold stimuli are released together," thereby producing these paroxysms of abdominal pain. It should be emphasized that the mesentery itself may be the source of pain. Distension by stretching of the appendix or small bowel mesentery, created by the swelling lymph follicles, may produce symptoms of abdominal pain.

INDICATIONS FOR SURGERY

Acute appendicitis is a relative surgical emergency and should be met as soon as the patient is adequately prepared for surgery. Mesenteric adenitis or intra-abdominal lymphoid hyperplasia may or may not be a surgical problem. If such a diagnosis can be clearly established, it should be treated medically with the physician being cognizant of the fact that, occasionally, appendiceal lymphoid hyperplasia will cause the obstruction of the appendiceal lumen and "classical" acute appendicitis results. It is our opinion that the analogy of appendiceal lymphoid hyperplasia to that of tonsilar hyperplasia is a useful corollary. The concept of the appendix as the "tonsil of the abdomen" is a valid one. Viral diseases and allergies are clearly established etiologic agents for lymphoid hyperplasia. For the child with an occasional attack of this disorder, there is no need to remove the appendix, any more than for the child with an occasional attack of tonsillitis to be subjected to tonsillectomy. However, the patient with intra-abdominal lymphoid hyperplasia producing symptoms on a recurrent basis should have the appendix removed.[2] It is assumed, of course, that such patients have been examined for the possibility of other causative factors and primarily for intestinal parasites. Such examinations require not only stool study but also night search for pinworms and the Scotch tape test. Recent one-day therapy for the protean pinworm infestation would seem empirically indicated in some cases. Anti-allergic type drugs should be administered to reduce hyperplasia of lymphoid tissue secondary to allergies. Often in my practice the initial one or two attacks of this syndrome are treated with antihistamine type drugs and adrenocorticotrophic hormone for its lymphopenic effect. Most patients respond well to this treatment. On the other hand, characteristic of the disease, is the fact that there are self-induced remissions. Should the patient, after two definite attacks of intra-abdominal lymphoid hyperplasia, appear with a third attack, it is recommended that interval appendectomy and complete exploratory laparotomy be performed. It is our opinion that with the increased incidence of viral infection there will be a greater incidence of intra-abdominal lymphoid hyperplasia. Our statistics have shown that we may continue to expect a greater incidence of appendices with lymphoid hyperplasia, having produced acute and subacute abdominal symptoms, to be removed.

SUMMARY

It is recommended that patients, having received proper medical therapy, who still persist

with several recurrent attacks of intra-abdominal and appendiceal lymphoid hyperplasia be given the benefit of an exploratory laparotomy with appendectomy to reduce morbidity and mortality from appendiceal disease.

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HAMARTOMA OF THE LUNG: REPORT OF SIX CASES

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Albrecht,[1] in 1904, first introduced the term "hamartoma" to designate a tumor-like formation consisting of an abnormal mixture of the histologic components normally encountered in an organ.

Until relatively recently, many cases of hamartomas were reported as chondromas; today, however, the term of hamartoma has been accepted universally.

It is a benign growth which occurs more frequently in men than in women and can be found in all age groups.

The lesion, of spherical shape in the majority of cases, is usually an incidental finding during a routine chest x-ray examination of an asymptomatic individual.

It is often located in the periphery of the lung. However, intraparenchymal, hilar as well as endobronchial, hamartomas are encountered, causing symptoms of compression and bronchial obstruction.

The roentgenogram usually shows a solitary, well circumscribed lesion which could very easily be confused with a bronchogenic carcinoma, or solitary metastasis, a solitary cyst, fungus granuloma or tuberculoma.

The tumor grows slowly, rarely exceeding 4 to 5 cm. in diameter; rare cases of large hamartomas, however, have been reported in the literature.[5] Busse[2] reported a huge hamartoma almost filling the entire hemithorax.

On gross examination and section the tumor has a characteristic white nodular surface; this appearance suggests the correct diagnosis with relative certainty.

Histologically, the cartilaginous element is predominant; areas of calcification or even ossification may be present.

The lesion is benign, well demarcated from the surrounding lung parenchyma, permitting in many instances its excision *in toto* by enucleation.

Cavin and associates[3] reported a case of malignant hamartoma and Davidson and Stern[4] presented a case with independent coexistence of hamartoma and bronchogenic carcinoma in the same lung.

Exploratory thoracotomy with excision biopsy is imperative for the establishment of definite diagnosis.

Local excision of the lesion by enucleation or wedge resection is usually adequate surgical treatment; however, an intraparenchymal or endobronchial location may necessitate more extensive resection or bronchoplastic procedures.

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CASE REPORTS

Case 1 (*4918*). L. G., a 62-year-old white man, was hospitalized because of a mass in the right midlung field, discovered during a routine chest x-ray survey (fig. 1).

An exploratory thoracotomy was performed, and a tumor of about 3 cm. in diameter was removed by enucleation from between the upper and middle lobe.

The tumor was sectioned and presented a whitish appearance compatible with hamartoma; this diagnosis was confirmed by microscopic sections, which disclosed a central mass of relatively adult cartilage, without any evidence of malignancy.

A parasympathectomy was also performed because of advanced pulmonary emphysema.

The patient had an uncomplicated postoperative course and uneventful recovery; he was discharged on the 11th postoperative day.

Case 2 (*4760*). M. R., a 61-year-old white housewife, was hospitalized because of an asymptomatic spherical lesion discovered incidentally in the left upper lobe (fig. 2).

A left thoracotomy was performed and a firm mass was found in the central portion of the left upper lobe; a left upper lobectomy was accomplished.

The mass about 3 by 2 cm. was lobulated. The cut surface varied from an opaque chalky white to a translucent blue-gray color. Microscopic sections revealed the presence of cartilage without any evidence of malignancy. The diagnosis of hamartoma was thus established.

The patient recovered satisfactorily and was discharged from the hospital on the 12th postoperative day.

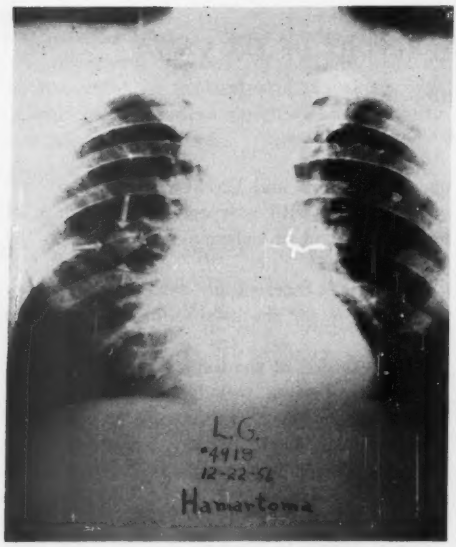

FIG. 1. Case 1 (4918). Spherical mass in right midlung field.

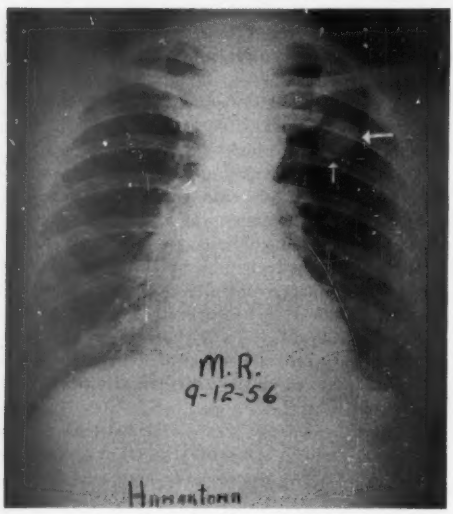

FIG. 2. Case 2 (4769). Round lesion in left upper lobe.

Case 3 (*4422*). K. V., a 64-year-old white man, was hospitalized because of a "coin" lesion in his left lung, discovered incidentally (fig. 3). He was completely asymptomatic.

Left exploratory thoracotomy was performed, which disclosed the presence of a mass in the superior segment of the left lower lobe.

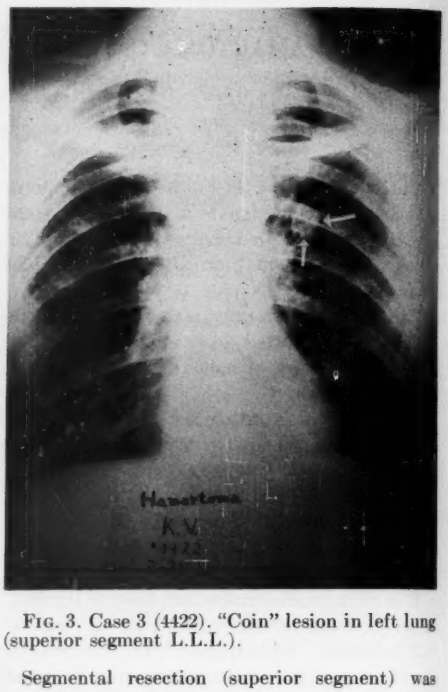

FIG. 3. Case 3 (4422). "Coin" lesion in left lung (superior segment L.L.L.).

Segmental resection (superior segment) was carried out, and frozen sections were obtained which confirmed the diagnosis of benign cartilaginous tumor, hamartoma.

The patient made an uneventful recovery.

Case 4 (*6013*). E. B., a 59-year-old white man, presented with an incidental finding of a small spherical lesion in his left lower lobe.

On exploration a small subpleural mass of 2 cm. in diameter was removed from the left lower lobe by wedge resection.

On microscopic sections the lesion proved to be benign connective tissue tumor compatible with hamartoma.

The patient was discharged on the 9th postoperative day.

Case 5 (*3289*). H. M., a 54-year-old white woman, was hospitalized with an asymptomatic spherical lesion, discovered during a routine chest x-ray.

A right exploratory thoracotomy was performed and a round firm mass was palpated in the periphery of the right lower lobe. This lesion was excised *in toto* by wedge resection.

It was an encapsulated mass, measuring approximately 2 by 2 cm., extremely firm and pale. Microscopic sections revealed several islands of cartilaginous tissue among epithelial and supportive elements.

Postoperatively she developed a cerebral vascular accident, with marked weakness of the entire left side.

She recovered and was discharged from the hospital on the 15th postoperative day.

Case 6 (4005). G. L., a 46-year-old white man, was admitted to the hospital with an incidental finding of a spherical lesion in his left lung.

Exploratory thoracotomy was performed and a small peripheral mass was excised by wedge resection from the anterolateral surface of the left lower lobe.

The cut surface of the specimen was glistening white in appearance, suggestive of chondroma. Frozen sections revealed lobulated masses of cartilage surrounded by connective tissue, confirming the benign nature of the lesion.

The patient was discharged on the 7th postoperative day.

SUMMARY

The results of treatment of six patients with hamartoma of the lung are presented.

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MULTIPLE STERCORACEOUS ULCERS OF THE COLON ASSOCIATED WITH HUGE FECALOMAS AND PERFORATION: CASE REPORT

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Perforations of the intestinal wall may be caused by many pathologic conditions. One of the rarer causes is the so-called stercoral ulcer. A stercoral ulcer may be defined as an ulcer produced by pressure from a hard, scybalous mass that collects most often in the large intestine.[1] An excellent study recently by Grinvalsky and Bowerman[2] reveals the paucity of this pathologic entity. Further study of the literature does not reveal any previous case of a perforated stercoraceous ulcer with generalized peritonitis and recovery. This is the report of such a case.

CASE REPORT

Patient was admitted to the hospital on February 12, 1960, for the third admission. He was a 55-year-old white man, admitted with the complaints of vomiting blood, dark stools and epigastric distress. His first admission to the Medical Center was in 1957 when he complained of some dysphagia. Entire work-up at that time was completely negative and the diagnosis was esophageal spasm. The second admission was in 1958 when he was admitted complaining of difficulty in swallowing. Again a complete gastrointestinal x-ray was done, an esophagoscopy was performed and there were no positive findings.

The present admission was characterized by an onset of epigastric distress for two to three months before the actual acute episode. The distress appeared mostly after meals and was relieved by simple medications. One hour prior to admission, the patient stated that he vomited approximately one quart of dark coffee ground material and passed two black stools, approximately 15 minutes apart. Following this he felt somewhat dizzy and was soon, thereafter, brought to the hospital. He had had no previous similar episodes.

Patient is married; his occupation is that of an electrician; and he gives a long history of drinking eight cans of beer per day and probably some hard liquor. The family history was noncontributory, as was history by systems.

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Physical examination at the time of admission showed patient's blood pressure to be 120/88; pulse, 108. He was well developed, moderately well nourished, appeared to be weak and lethargic, but was oriented. Head and neck were negative; chest, symmetrical; diameter of the heart, normal; lungs, clear; liver, palpable two to three fingers below the costal margin; spleen, not palpable. There was mild tenderness to palpation in the epigastrium; the genitalia were normal; the extremities were not remarkable, and the neurologic examination was negative. Laboratory investigations showed a hemoglobin of 16.1 gm.; hematocrit, 47 per cent; leukocytes, 14,400 with a normal differential. Urine showed a few white cells and red cells. Upper gastrointestinal x-ray showed a moderate size sliding type hiatus hernia with ulceration of the mucosa. The impression at the time of admission was gastrointestinal hemorrhage due to either a hiatal hernia or esophageal varices and fatty infiltration of the liver due to alcoholism.

The patient was treated conservatively with bedrest, antacids and Sippy diet. He got along fairly well except for some occasional low cramping abdominal pain. On February 17, 1960, a barium enema showed multiple diverticula in the sigmoid, but due to an excessive amount of fecal material in the colon a second examination was advised. Diagnosis was deferred at this time.

The following day, on February 18, 1960, the patient suddenly became markedly distended with difficult and labored breathing. Physical examination showed an acutely ill patient with what appeared to be generalized peritonitis. The abdomen was tremendously distended and tender throughout. There was slightly more tenderness in the right lower quadrant. Provisional diagnosis was a perforated appendix with generalized peritonitis. However, when the flat plate of the abdomen was taken, the retained barium, from the earlier enema, showed that the appendix had filled, and had remained filled, with barium (fig. 1). Therefore, the presumptive diagnosis of ruptured appendix was ruled out. At the same time, the flat x-ray film showed slight distension of the small intestine and a tremendous distension of the

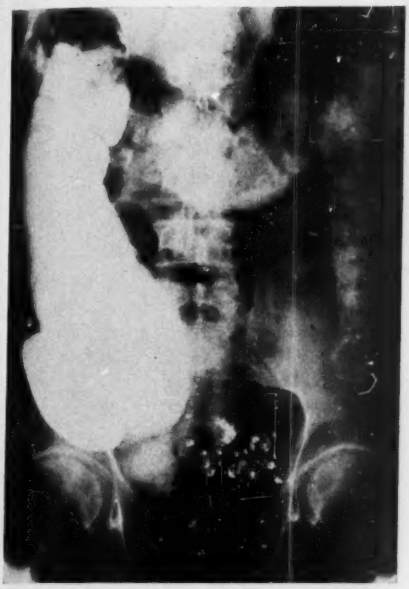

FIG. 1. Barium enema revealing marked distension of the cecum and abrupt termination of barium on left outlining fecaloma.

cecum. At this point a preoperative diagnosis of large bowel obstruction was made, the patient taken to surgery and, under general anesthesia, a cecostomy for relief of the large bowel distension was performed. Before the opening of the cecum and the insertion of the catheter, a culture was taken from the peritoneal cavity which later showed *Streptococcus viridans* contamination of the peritoneal cavity.

The patient progressed moderately well the next day, but the following day he became maniacal and his condition became worse. He still had marked abdominal distension, and another flat plate showed a paralytic ileus. It was difficult to control his maniacal symptoms. Because of a marked cerebral irritation it was decided to place the patient in hypothermia. Following intravenous curare to control the shivering, he was placed in hypothermia and his temperature lowered to 90 degrees. Then he was given 500 cc. of whole blood. His fluid and chemical balance were corrected. Finally after large doses of intramuscular paraldehyde heu qieted down. A flat x-ray taken at this time revealed an increase in the degree of distension since the previous operation. Negative suction was instituted at this time, but afforded little relief.

On February 22, 1960, he improved although there were still no bowel sounds present, and another flat plate revealed continuing distension of the small bowel. At this time he was removed from hypothermia with some improvement. The negative suction was working well; the cecostomy was draining well. The flat plate taken on this date revealed that the barium from the original barium enema was still present as far down as the distal sigmoid, beyond which point there was no barium. This was interpreted to be a stricture of the colon due to diverticulitis and retention of fecal material.

The patient progressively improved for the next 10 days. The paralytic ileus diminished, the cecostomy functioned well; and he was able to be up and around, but during this time he began to develop a large mass in the left lower quadrant. Since the distension had subsided, one could at this time feel a mass of fist size in the left lower quadrant. It was then surmised that he had a ruptured diverticulum with abscess formation.

On March 1, 1960, the patient was taken to surgery, a proctoscopic examination was performed and an attempt to aspirate and drain the abscess through the rectum was made, but this was unsuccessful. The patient continued to improve and on March 15, 1960, was again taken to surgery, where under general anesthesia he was explored. The sigmoid was found to be very distended and within it there were two very hard

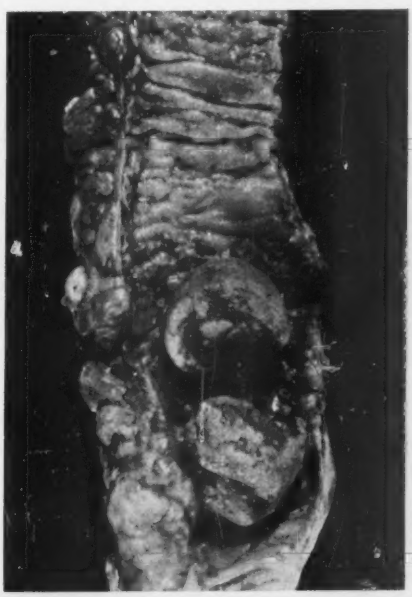

FIG. 2. Opened resected portion of colon revealing huge fecalomas.

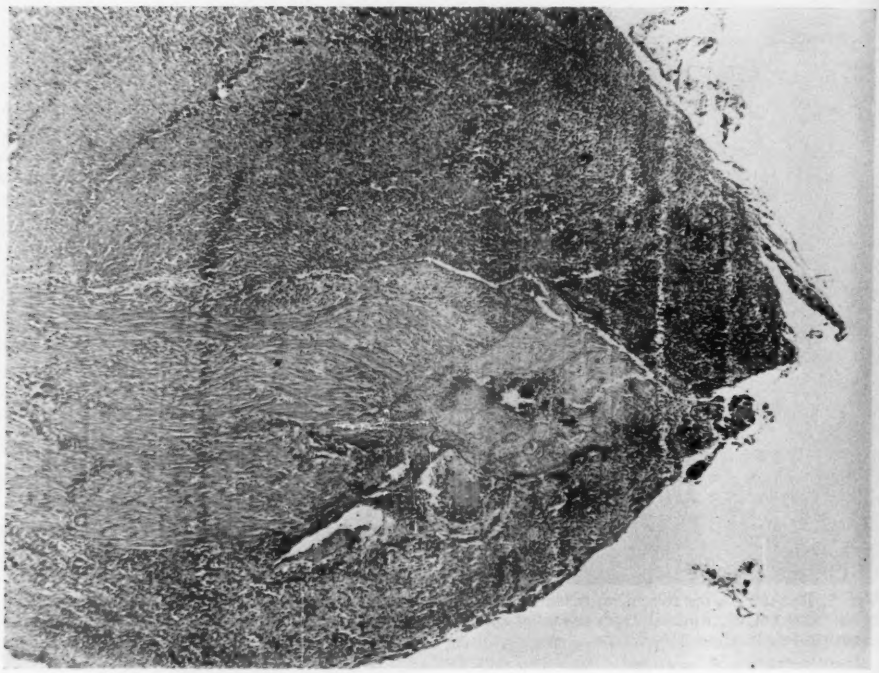

FIG. 3. Photomicrograph ×23 showing tract of perforation to the right

masses. A perforation on the anterior surface was present directly over the hard mass. There was a tremendous amount of inflammatory reaction about the bowel, but the rectal portion of the descending colon was normal.

A colon resection was performed starting approximately from the transverse colon, continuing distally to the sigmoidorectal junction, and an end-to-end anastomosis between these two areas performed. After resection, and after opening the colon, two huge and very hard fecalomas, each measuring about 10 cm. in diameter, were found. Beneath the base of one of these was a perforated stercoraceous ulcer of the sigmoid colon.

PATHOLOGY

The resected specimen was 40 cm. in length containing two large, very firm masses of brownish-green fecal material. These masses were hard and measured 3.5 by 4.0 cm. The dilated portion of colon showed many ulcers, some of which measured up to 4.0 to 5.0 cm. in greatest dimension. The perforation appeared 15 cm. from the distal line of resection. The areas of ulceration elsewhere in the colon varied in extent from those which involved only mucosa to those which extended almost through the muscularis (figs. 2–4).

DISCUSSION

Fecal stones or fecalomas may be caused by several factors, such as mechanical obstruction to the normal outflow tract or any incomplete obstruction which may delay the passage of fecal material and cause it to form a hard fecal ball. If the normal amount of mucus is diminished, the feces may become hard and dry and concretions may form. Megacolon may be a predisposing factor. Occasionally the nucleus of a fecaloma may be a gallstone. In the aged, bedridden patient with poor bowel habits, concretions may occur. A barium enema, with imperfect emptying of the bowel, so that the barium coats the fecal material, may also cause the same condition. Foreign bodies, such as nails, chicken bones and other such objects, may be the nidus of a fecaloma. Stricture of the colon, following acute inflammatory diverticulitis, may also slow the fecal stream and produce fecalomas.

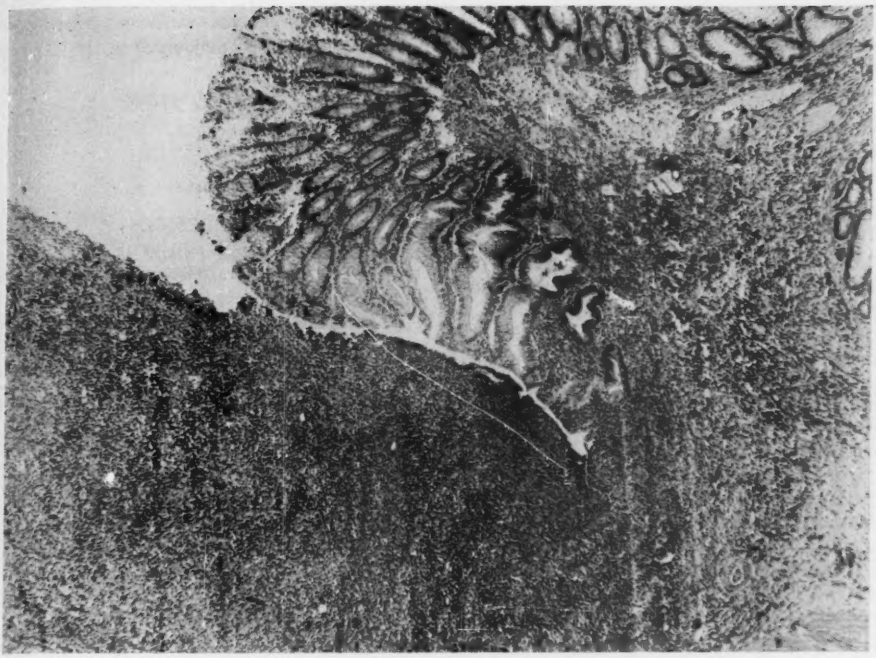

FIG. 4. Photomicrograph ×23 showing junction of stercoral ulcer with intact mucosa

SYMPTOMS

Prior to the time a fecaloma causes enough pressure to produce a perforation through a stercoraceous ulcer of the colon, the symptomatology is usually that of a low grade intestinal obstruction. However, this is infrequent because the fecalomas slide up and down the bowel wall and allow some feces to pass distally. When the actual perforation occurs, it is the same type of catastrophe that occurs any place in the peritoneal cavity when a viscus has perforated.

The patient exhibits signs of shock, a rapid, thready pulse, increasing abdominal distension, generalized rigidity and tenderness. Flat x-ray examination of the abdomen may show increasing distension of the large bowel, and if the distension is marked and obstruction evident, bowel decompression is indicated.

Fecalomas are most common in the rectosigmoid area, but they may occur in the cecum, the transverse colon, the appendix and the small intestine. In the small intestine they are most often located in the ileum about 5 to 10 inches from the ileocecal valve. The size may vary from small multiple fecalomas to a huge mass. Usually there is a past history of constipation, or alternating constipation and diarrhea with intermittent cramping pains. The masses may be so large that they can press against the urinary bladder or prostate and produce a urinary obstruction. Occasionally there may be blood in the stools but this is not usual.

Complications of fecalomas are: (1) stercoraceous ulceration with perforation; (2) if it appears in the appendix it may produce inflammation like appendicitis or cause a cecal abscess.

TREATMENT

If the fecalomas are low in the rectum, it may be possible to break them up by digital manipulation, repeated oil enemas and any material that may soften the fecal mass. If they are higher in the colon it is difficult under any circumstance to attempt to reduce the size; however, all conservative treatment should be attempted. Mild laxatives by mouth, repeated oil retention enemas, or enemas with hydrogen peroxide, may help to break down fecalomas. If conservative

treatment is unsuccessful, surgery may be indicated.

The pathologic anatomy of a perforated stercoraceous ulcer is primarily due to direct pressure on the intestinal mucosa, which produces an ischemic necrosis and perforation.

The usual chemical material of a fecaloma has been described by Harland.[3] In his series he found that most of these concretions contain an average of 15.8 per cent calcium, 8.2 per cent phosphorus, and that the organic matter was composed of nitrogenous fecal debris.

SUMMARY

A case of perforated sterocoraceous ulcer of the colon associated with huge fecalomas and generalized *Streptococcus viridans* peritonitis has been presented. The difficulties in the diagnosis have been discussed.

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PENETRATING WOUNDS OF THE INFERIOR VENA CAVA

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Injuries to the inferior vena cava with survival are seldom reported in the medical literature. Such injuries are caused by: (1) penetrating wounds; (2) external (nonpenetrating) wounds; and (3) injury incurred during surgical procedure. Regarding the first two categories of injuries, Kidd[1] in 1945 reported a collected series of ten cases, with survival in eight, including one patient of his own. In 1957 Starzl and co-workers[2] reported three gunshot wounds of the inferior vena cava successfully treated, and stated that these represent the eighth, ninth and tenth cases of missile injury to the inferior cava in the literature with survival. The latter authors also collected seven cases reported as surviving stab wounds or blunt trauma lacerations of the inferior vena cava.

A report of an additional patient with repair and survival after gunshot wound of the inferior cava follows.

CASE REPORT

A 17-year-old boy entered Good Samaritan Hospital, Phoenix, Arizona, at 10:30 p.m. on February 9, 1959, after being shot in the abdomen a half-hour previously by a .22 caliber bullet. He walked a distance of several blocks unassisted after the injury and became weaker, sweaty, cold, thirsty and vomited.

Past history was one of excellent general health except for obesity.

Admission examination disclosed a pale, obese young man in mild stupor, with moist skin and uncoordinated eye movements. Pulse, 108 per minute; blood pressure, 64/40. The abdomen was large and round with a penetrating wound of entrance in the left epigastrium, generalized tenderness and hypoactive peristalsis. No wound of exit was found. The lower extremities had normal motor and sensory findings.

Initial treatment included Trendelenburg position, warm blankets, 1000 cc. of 6 per cent dextran and 500 cc. of whole blood which corrected the clinical shock findings; then the patient was taken to the operating room. Through a left upper paramedian incision, exploration revealed a massive hemoperitoneum, a large retroperitoneal hematoma and numerous perforations of the large and small bowel, but no definite source of bleeding. The incision was extended down to the pubic bone. The mobile portions of intestine were packed outside of the peritoneal cavity and a perforation was seen in the posterior parietal peritoneum. The posterior peritoneum was opened longitudinally and bleeding was controlled by direct digital pressure over a gauze pad. Locating the injured portion of vena cava was difficult in the large retroperitoneal hematoma until dissection was started over the right iliac vein and iliac artery. With these structures well delineated, the dissection proceeded readily in a cephalad direction along the vena cava until anterior and posterior perforations were found in the cava. These were each repaired with running sutures of 5-0 arterial silk, after applying proximal and distal compression to the vena cava. Thereafter, with the patient's condition stabilized, the intestinal wounds were evaluated and nine perforations (one was tangential) were treated by repair or resection. A total of 1000 cc. of dextran and 3000 cc. of whole blood was given in the preoperative and operative periods.

The postoperative convalescence of this patient was uneventful and he has made a complete recovery.

Patients having wounds of the inferior vena cava, who are alive on reaching the hospital, have the benefit of tamponade effect in the retroperitoneal area to prevent death due to exsanguination. It is of prime importance during surgical treatment of patients with retroperitoneal hemorrhage in whom major vessel injury is suspected, to avoid opening of the posterior parietal peritoneum and releasing of the tamponade control until adequate preparation is made to replace rapid blood loss and to handle the vascular injury. An injured vena cava surrounded by a large hematoma is more difficult to approach surgically than is the aorta (fig. 1), because the vein has thin, readily collapsible walls compared to the thicker, sturdy walls of the aorta, which are easily identified by palpation and visualization during the course of a dissection.

In this patient we found it to be quite advan-

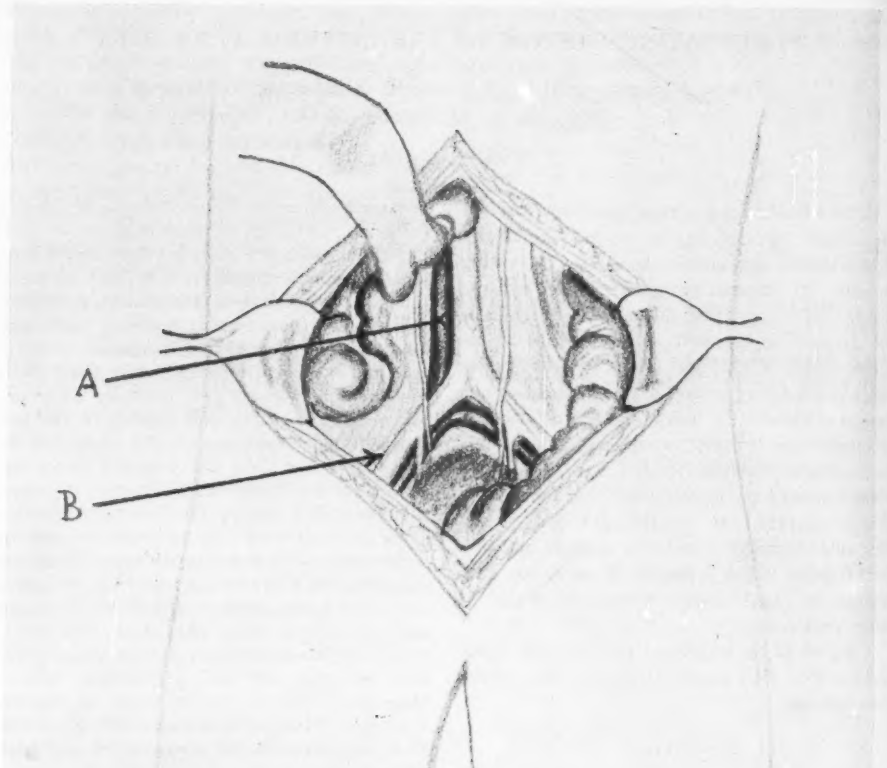

FIG. 1. Diagram of abdominal structures showing: *A*, the site of perforating wound in inferior vena cava producing a massive retroperitoneal hematoma, and *B*, the area where dissection of the retroperitoneal structures should be started along the iliac vessels up through the hematoma to the site of the caval perforation.

tageous to control the caval bleeding by direct manual compression while dissection was started along the right iliac artery and vein. The dissection was then carried up the inferior vena cava to the sites of injury allowing anatomic accuracy in handling a damaged vein surrounded by a large hematoma.

SUMMARY

A report is given of a patient with perforation of the inferior vena cava and intestine with repair and recovery. Significant points in management of penetrating wounds of the inferior vena cava are avoidance of opening the retroperitoneal hematoma until preparations have been made to control and replace rapid blood loss, and starting dissection of the venous system at a distance from the site of injury to permit anatomic clarification of this delicate structure.

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INVESTIGATIONS REGARDING THE USE OF ANTIBIOTICS IN STRANGULATION OBSTRUCTION*

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Strangulation of a short segment of ileum in experimental animals is followed by a rapid downhill course, and death usually occurs within 24 to 30 hours.[5] Postmortem examination reveals that the peritoneal cavity contains from 100 to 400 cc. of dark, foul fluid. When the segment of strangulated bowel is placed in a plastic bag so that exposure to this fluid is prevented, the life of the animal is indefinitely prolonged.[7] The dark fluid has been shown to be lethal for normal dogs in amounts as small as 2 cc. per kg. when injected into the peritoneal cavity. The conclusion that this material plays a major role in the lethal issue of strangulation obstruction cannot be escaped. Previous studies have shown that bacterial cells are primarily responsible for the toxic properties of peritoneal fluid resulting from strangulation obstruction.[1, 2] When the segment of strangulated bowel is filled with certain antibiotics, the resulting peritoneal fluid has been found to be nontoxic. Antibiotics also exert a measure of protection for normal animals following exposure to toxic strangulation fluid.[3] The following experiments are concerned with the efficacy of various antibiotics as well as the routes of their administration.

MATERIALS AND METHODS

Normal adult mongrel dogs were used for the following experiments.

Donor animals (31 dogs). With intravenous pentobarbital anesthesia and sterile technique, the abdominal cavity was opened in the midline. A closed loop obstruction of the ileum was created by passing umbilical tape ties around each extremity of a 10-cm. segment. This portion of the bowel was then strangulated by division and ligation of its venous drainage.[4] The animals were then placed in their cages where they remained until death occurred. The fluid which had accumulated in the peritoneal cavity was withdrawn, placed in sterile containers and refrigerated until used.

Recipient animals. Previous studies have shown that the intraperitoneal injection of toxic strangulation fluid was attended by considerable pain for the unanesthetized animal. For this reason all animals were lightly anesthetized with intravenous pentobarbital before the fluid was injected.

Series I (11 dogs). These animals served as controls and were used to determine the lethality of various aliquots of strangulation fluid. Each animal was given an intraperitoneal injection of strangulation fluid in the amount of 3 cc. per kg.

Series II. The purpose of the following studies was to determine the efficacy of various routes of antibiotic administration following exposure of normal dogs to toxic strangulation fluid. All animals were given an intraperitoneal injection of strangulation fluid in the amount of 3 cc. per kg. The antibiotic was administered immediately after the fluid had been given.

Group 1 (20 dogs). These animals were given penicillin (100,000 U. per kg.) and streptomycin (0.125 gm. per kg.). The antibiotics were injected into the peritoneal cavity of 10 animals while the remainder received an intramuscular injection.

Group 2 (40 dogs). Chloramphenicol in the amount of 50 mg. per kg. was given to all animals of this group. It was injected intramuscularly (10 dogs), intravenously (mesenteric vein) (10 dogs), intravenously (femoral vein) (10 dogs) and intraperitoneally (10 dogs).

Group 3 (30 dogs). These animals were given kanamycin (50 mg. per kg.). It was injected into the muscle (10 dogs), the femoral vein (10 dogs) and the peritoneal cavity (10 dogs).

Series III (30 dogs). The object of these studies was to compare the efficacy of various antibiotics when given along with larger doses of strangulation fluid (6 cc. per kg.). The antibiotics and the strangulation fluid were given intraperitoneally.

Group 1 (10 dogs). Penicillin (100,000 U. per kg.) and streptomycin (0.125 gm. per kg.).

* From the Department of Surgery, University of Mississippi Medical Center, Jackson, Mississippi. Supported by United States Public Health Service, grant A-4644.

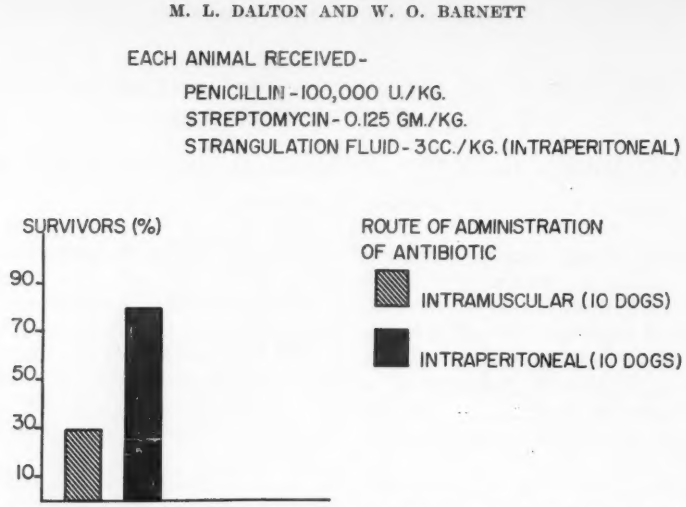

FIG. 1. The efficacy of various routes of administration of penicillin-streptomycin in animals exposed to strangulation fluid.

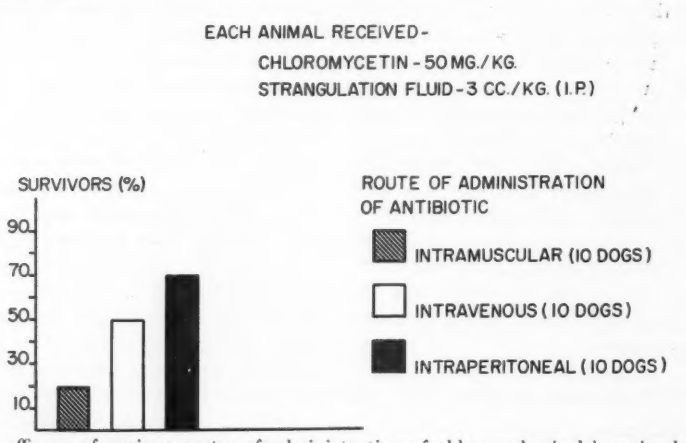

FIG. 2. The efficacy of various routes of administration of chloramphenicol in animals exposed to strangulation fluid.

Group 2 (10 dogs). Chloramphenicol (50 mg. per kg.).

Group 3 (10 dogs). Kanamycin (50 mg. per kg.).

Series IV (30 dogs). These animals received the larger dose of strangulation fluid followed by massive doses of antibiotics.

Group 1 (10 dogs). Penicillin (300,000 U. per kg.).

Group 2 (10 dogs). Chloramphenicol (200 mg. per kg.).

Group 3 (10 dogs). Kanamycin (200 mg. per kg.).

Series V (10 dogs). These animals received an intraperitoneal injection of strangulation fluid in the amount of 6 cc. per kg. By the same route of administration they were also given kanamycin (200 mg. per kg.) and penicillin (300,000 U. per kg.).

RESULTS

Donor animals. These dogs lived from 20 to 32 hours. Their postoperative course was not unusual until about 2 hours before death, when they rather suddenly developed rapid respiratory rates, became lethargic and then coma-

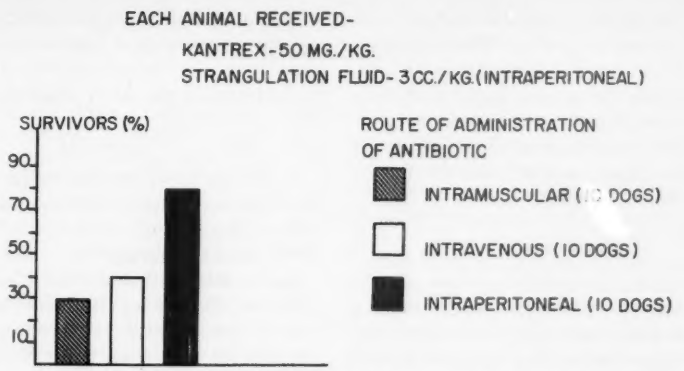

FIG. 3. The efficacy of various routes of administration of kanamycin in animals exposed to strangulation fluid.

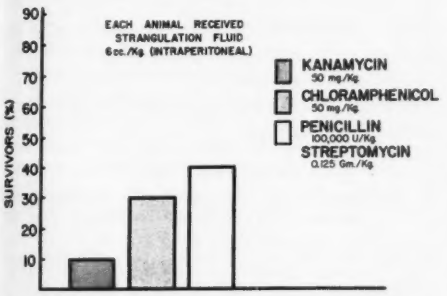

FIG. 4. Results with increased doses of strangulation fluid.

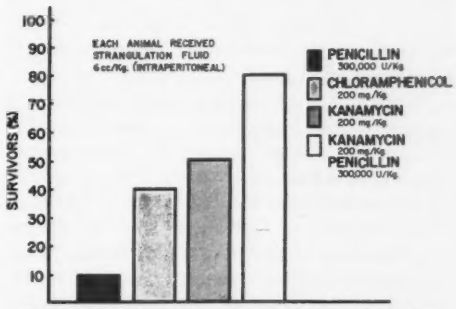

FIG. 5. Results with increased doses of strangulation fluid and antibiotics.

tose. From 100 to 400 cc. of dark, foul fluid was removed from the peritoneal cavity of each animal. Although there were usually no areas of gross perforation, the obstructed intestinal segments were collapsed.

Recipient animals. Many of the animals which failed to recover never regained consciousness following exposure to strangulation fluid. Some of the animals awoke but appeared to be progressively lethargic until they expired. When adequate antibiotic coverage was afforded, animals recovered rapidly from anesthesia, moved about their cages normally and survived. The number of 24-hour survivors was recorded for each group.

Series I. All control animals expired within 24 hours except one which lived for 70 hours.

Series II. Five of the 10 animals which received an intramuscular penicillin-streptomycin injection recovered. When these antibiotics were given intraperitoneally, 9 of 10 animals recovered (fig. 1). Following intramuscular injection of chloramphenicol, 2 of 10 animals recovered but the intraperitoneal administration of this antibiotic resulted in the survival of 7 of 10 animals. When chloramphenicol was injected into the superior mesenteric vein, 4 of 10 animals recovered. There were 5 survivors among the 10 animals that received chloramphenicol by way of the femoral vein (fig. 2). When kanamycin was given intramuscularly to 10 animals there were 3 survivors. Four of 10 animals recovered after the intravenous injection of kanamycin. The intraperitoneal injection of this antibiotic in 10 animals resulted in 8 survivors (fig. 3).

Series III. The survival rates were much lower in this series because of the larger dose of strangulation fluid. There were 4 survivors among the 10 animals which received penicillin and streptomycin. Following the administration of kanamycin there was 1 survivor and 3 dogs recovered after the injection of chloramphenicol (fig. 4).

Series IV. Among the 10 animals that received penicillin there was 1 survivor. When kanamycin was given there were 5 survivors and following the administration of chloramphenicol there were 4 survivors (fig. 5).

Series V. The combination of penicillin and kanamycin in large amounts resulted in the recovery of 8 of 10 animals.

DISCUSSION

Antibiotics afford greater protection for experimental animals following exposure to toxic strangulation fluid than any other agent. Previous studies have shown that recipient animals experience a severe leukopenia as a result of the intraperitoneal injection of toxic fluid.[6] The fluid contains large numbers of bacterial cells of many varieties. These organisms may be cultured from the blood stream following exposure to strangulation fluid.

Support of body mechanisms by antibacterial agents at this time of severe stress probably accounts in a large measure for the improved survival rates found in antibiotic-treated animals. Best survival rates resulted from the intraperitoneal injection of antibiotics. This was true for all antibiotics used in this study. Mixing of the antibiotics with the strangulation fluid in the peritoneal cavity may account for this difference. Intraperitoneal antibiotics have been used in the treatment of 10 patients with strangulation obstruction. Kanamycin (0.5 gm.) dissolved in 100 cc. of saline was left within the peritoneal cavity after thorough irrigation had been carried out. No complications relating to this antibiotic in the dose mentioned above have been noted. In experimental animals the intravenous route of antibiotic injection gave better results than intramuscular administration. There was no apparent advantage afforded by the intraportal injection of chloramphenicol. It appeared that combination of antibiotics gave better results than single administration of these agents. With the smaller antibiotic dosage the combination of penicillin and streptomycin gave best results. When large doses were used better survival rates were obtained following the use of penicillin and kanamycin.

SUMMARY

1. Antibiotics afford significant protection to experimental animals following exposure to the toxic peritoneal fluid which results from strangulated intestinal obstruction.

2. The intraperitoneal route of administration was most effective for all antibiotics used.

3. Better survival rates resulted from the intravenous than from the intramuscular injection of antibiotics.

4. The combination of penicillin and kanamycin gave best results following exposure to large doses of strangulation fluid.

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Book Reviews

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Surgical Treatment of Unequal Extremities. By CHARLES WEER GOFF, M.D. Charles C Thomas, Springfield, Ill., 1961.

The author's text long has been needed within the field of orthopedics to further define and explain the problem of unequal extremities. It is sufficiently straight forward so that the less experienced might read the text with understanding, and yet sufficiently complete so that an experienced orthopedist can gain insight and background in handling his own cases.

The subject is introduced with a chapter on the growth of long bones that contains a good review of methods of measuring the extremities. Bone shortening and lengthening are dealt with in a separate chapter, with worthwhile statements of preference on the author's part. The subject of stapling as well as epiphysiodesis is dealt with thoroughly, and photographs demonstrating clinical results are included in the text. Of particular interest to the clinician is the chapter on long bone fractures and growth.

A. GIBSON PACKARD, JR., M.D.

Instructional Course Lectures, 1960. Edited by FRED C. REYNOLDS, C. V. Mosby Co., St. Louis, 1960.

This year's (1960) *Instructional Course Lectures* of the American Academy of Orthopaedic Surgeons is divided into five interesting parts.

Part One deals with Fractures. Consideration is given, initially, to fractures about the elbow in children. Thirty plus well written pages are devoted to the care, management and pitfalls of these injuries. The article is crystallized by the author's fourteen paragraphs in the summary and lends considerable insight to the neophyte's management of these problems.

The following two articles deal with the diagnosis and the management of arterial injuries. The contemporary diagnostic measures, techniques and case reports are presented.

Intense interest in the hip is demonstrated in a discussion of management of the ununited fractures of the neck of the femur followed by a symposium on the management of fresh fractures of the femoral neck.

In the symposium Doctors Cave, Green, Badgley, Soto-Hall and Hinchly discuss the many aspects of the femoral neck fracture. Manipulations, operative technique, complications, replacement prostheses, various means of fixation, causes of failure and pages of photos and tables are presented. The current thinking and trends in this captivating problem are condensed into these six articles, the authors frequently coming into agreement on basic problems and concepts, but occasionally one or two sailing off on a slightly different view point to deal with an aspect which to that individual is pivotal.

As an example Doctor Green believes union is most dependent on extremely sound mechanical fixation. This is accomplished by a Smith-Peterson nail and side plate accompanied by four Moore pins in the same hip.

Doctor Badgely places more stress on the sliding nail techniques, which allow continued contact, continuity and compression of the fragments with stability.

Thus the hip fracture remains a problem, but the discussions reflect current thinking and make excellent sources of reference.

Part Two is devoted to Bone Graft Surgery. A very worthwhile symposium describing techniques, indications, experimental and clinical applications is delivered. This is followed with a sojourn into non-union, causes and treatment with a review of 741 cases. The fate of grafts and a survey of research in the past decade is presented.

Part Three is devoted to Children's Orthopaedics. It begins with a discussion of "Skeletal Age and the Control of Bone Growth." Observations of patterns of growth in different individuals are compared with the mean and the methods of assessment of skeletal maturity are described. The value of growth prediction charts in planning epiphyseal arrests is emphasized followed by a discussion of the problems in correction of discrepancies.

Illustrative cases are presented and the need for proper timing of the operative procedure in accordance with the individual variation of each patient is re-emphasized.

The next article, "Unequal Leg Length" by Doctor Blount, points up the need to seek out secondary and functional causes for shortening and/or lengthening, as well as the actual shortening. The need for initial correction of the "secondary factors," such as tight abductors of the hip,

tight, weak, strong or contracted "hip shruggers," is emphasized. Then an assessment of the actual shortening may be made.

In summary the lecture series cannot be recommended too highly. The orthopaedic resident should not miss reading the volume word for word. The practicing orthopaedist and traumatic su geon also will find the material extremely valuab The volume is quite attractive, the paper of exc lent quality and the amount of factual informati based on the experiences of the country's forem authorities makes the reading a pleasant necessit

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